Studies on maydis leaf blight (Drechslera maydis) of fodder maize (Zea mays L.)

THESIS

Submitted to the

Bundelkhand University, Jhansi

For the award of

DOCTOR OF PHILOSOPHY

In

BOTANY

By

PRAVEEN KUMAR

Under the Guidance of

Dr. Pradeep Saxena

2006

Crop Improvement Division
Indian Grassland and Fodder Research Institute
JHANSI - 284003 (U.P.)

INDIAN GRASSLAND AND FODDER RESEARCH INSTITUTE
GWALIOR ROAD, JHANSI, 284003 (UP) INDIA

No. HRD/BU/05-06
7th June, 2006

Dr. K A. Singh
Director

To,
The Vice Chancellor,
Bundelkhand University,
Jhansi. (U.P.)

Subject: Forwarding of Ph.D. thesis in Botany (plant pathology)
Reference: B.U./ Adm. / Research / 2004 / 787-89 dated 30-10-2004

Sir,

I am forwarding herewith the thesis entitled **"Studies on maydis leaf blight (*Drechslera maydis*) of fodder maize (*Zea mays* L.)."** by Mr. Praveen Kumar for the award of degree of Doctor of Philosophy in Botany in Bundelkhand University, Jhansi. The work has been carried out under the supervision of Dr. Pradeep Saxena, Sr. Scientist (Plant Pathology) at this Institute.

Thanking you,

Yours Sincerely,
kasingh
(K.A. Singh)

Encl.: a/a

DECLARATION

I hereby declare that the thesis entitled **"Studies on maydis leaf blight (*Drechslera maydis*) of forage maize (*Zea mays* L.)"** being submitted for the degree of Doctor of Philosophy in Botany in Bundelkhand University, Jhansi (U.P.), is an original piece of research work done by me under the supervision of Dr. Pradeep Saxena, Senior Scientist (Plant Pathology) Indian Grassland and Fodder Research Institute, Jhansi (U.P.) and the best of my knowledge and belief, is not substantially the same as one which has already been submitted for a degree of any other academic qualification at any other University or examining body in India or abroad .

07/06/06

(Praveen Kumar)

INDIAN GRASSLAND AND FODDER RESEARCH INSTITUTE
GWALIOR ROAD, JHANSI, 284003 (UP) INDIA

Dr. Pradeep Saxena
M.Sc., Ph. D, FPSI, M.N.A.Sc.
Senior Scientist (Plant Pathology)
Crop Improvement Division

Tel: 91-0517-2447245 (R)
Fax: 91-0517-2730833
Gram: Ghasanusandhan
E-Mail: pradeepsaxena@lycos.com

CERTIFICATE

This is to certified that the thesis entitled " **Studies on maydis leaf blight (*Drechslera maydis*) of forage maize (*Zea mays* L.)**" being submitted by Praveen Kumar for the award of Ph.D. degree in Botany (plant pathology), Bundelkhand University, Jhansi, contains original piece of research work.

It is further certified that:

a. The thesis has been duly completed;

b. the thesis embodies the work of candidate himself, and

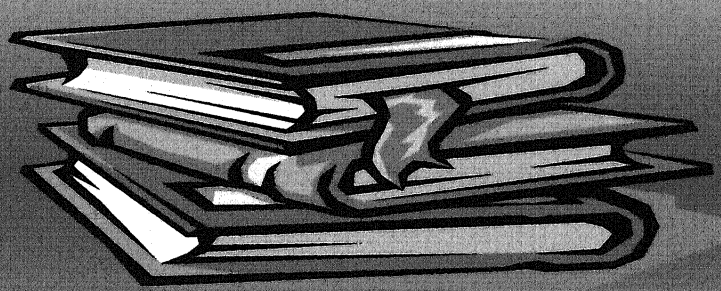
c. it is up to the standard both in respect of it's contents and literary presentation for being referred to the examiners.

It is also certified that the candidate had worked under my guidance and supervision for the period required under the University's Research Ordinance - 7. The candidate has put the required attendance in the Department during this period.

07.06.06

(Pradeep Saxena)

DEDICATED TO



My Parents &
Teachers

ACKNOWLEDGEMENTS

With sincerity and great pleasure. I acknowledge my heart felt gratitude to my adviser Dr. Pradeep Saxena, Sr. Scientist (Plant Pathology) Division of Crop Improvement. Indian Grassland and Fodder Research Institute Jhansi for his inspirational guidance, encouragement, Nobel behaviour, scholarly suggestion, advise, constructive criticism and never ending patience at each step of my Ph. D. work. Without his untiring effort, this work could not have been accomplished successfully with in time. There is no such words by which I am able to express my sincere thanks for this valuable helps and supports.

I am extremely thankful to Dr. K.A. Singh Director, and Ex-Director Dr. P.S. Pathak, Indian Grassland and Fodder Research Institute Jhansi for granting me the permission for higher studies and silent support and encouragement.

I am also very thankful to chairman and all member of H.E.C. of the institute for their continuous support and encouragement.

It gives me immense pleasure and privilege to express my sincere and deep sense of gratitude to Dr. G.P. Shukla Head of Division and Dr. R.N. Chaubey, Ex- Head of Division for providing the full facilities of my research work.

It is also my pleasure to pleasure to acknowledge the help received from the members of advisory board Drs. Amresh Chandra, R.K. Bhatt and R.B. Bhaskar.

I am also thankful to Dr. D.R. Malaviya for providing the maize genotype for my Ph.D. work.

I am also grateful to Dr. Sharmila Roy for their help in statistical analysis and interpretation. Which help me in completion of this work.

I offer my sincere thanks to Dr. S.N. Zadoo, Dr. N. Hasan, Dr. M.G. Gupta, Dr. Maharaj Singh, Dr. M.I. Azmi, Dr. K.C. Pandey, Dr. M.J. Baig, Dr. U.P. Singh & other scientist who provides me, needed help and moral support s for completing my Ph.D. work.

I am also extremely grateful to Dr. N.K. shah for his continuous support and encouragement.

Cordial thanks to staff members of plant protection laboratory viz. Shri P.K. Tyagi. (Technical Officer), Shri RamDin (Laboratory Assistant), Shri A.B.Panday (T-4) and Shri Virendra Kumar (Lab Attendant) for helping me during my lab work.

I offer my sincere thanks to Shri Ram Swaroop for the help in my experiment and data recording.

It was impossible for me to complete my work without the help of my colleagues, Dr. Yachna Mishra, Mr. B.M.S. Parihar, Mr. Pushpendra Kumar Verma, Ruquiya Bano, Mohani Ahirwar and Rashmi Nigam for providing moral support and ungraudging help.

I owe my thanks to my loving friends Dr. S.K. Srivas, Deepti Gupta, Kamna Gupta, Ankita Mukharji Vandana Khare Monika Singh and Mamta Rajpoot who always given me moral support in my all adds and inspired me to do work with a great vigour.

I express my sincere thanks to Mr. Rajeev Kumar Kushwaha, Mr. Karan Singh, Mr. Pushpendra Agnihotri and Mr. Nishil Kumar Jain research students for providing me help during my Ph.D. work.

Thanks are also due to Mr. G.P. Nigam (Library) and Mr. K.P. Rao (ARIS) for their timely help.

My thanks are also due to Mr. Jagdish Prasad and Mr. Dheeraj Kumar Dhingra for typing and formatting of the thesis.

Above all the whole credit for the completion of the work goes to my beloved parent Shri Dr. Jugal Kishor and Smt. Sheela Davi who constantly supported me during the course of investigation.

Words never can express but for very pleasure of it. I would like to extend my most sincere thanks to my uncle Shri K.R. Shashi, Shri Kalicharan and Shri Rajendra Kumar for encouragement every time of my Ph.D. work.

My elder brother Mr. G.P. Singh and Mr. Brajesh Singh provide me enormous strength and spare my self from domestic duties, I express my gratitude to them.

I am so thankfull to my well wishers Mr. Santosh Kumar, Mr. J.K. Amarya, Mr. Harish Khoiya, Dr. Aditya Verma and Mr. Kailash Chandra, Mr. Mohan Lal (Mama ji) for their moral encouragement.

I can not forget the sweet love of my sisters Mrs Asha, Mrs Anita Amarya, Mrs Gayatri Khoiya and Mrs Sunita Verma.

I can not also forget my cousin Sunil Kumar, Manoj Kumar, Mukesh Kumar, Ravi Kumar, Manish Kumar and Pankaj.

My sincere appreciations are also to my uncle Shri. ShriRam Sikaniya(Technical Officer) and Shri N.L. Sinoriya(AAO) whose guidance abounding love affection helped me in completion of my Ph.D work.

I must pay my thanks to all these unseen hands that cooperated in this work.

Last but not least I would like to thank to almighty God, without his generous blessing. I would not be able to complete this work.

CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Chapter -1

INTRODUCTION

Corn (*Zea mays L.*) originated in the western hemisphere, was the American Indian systematically cultivating the only cereal although some other grains were harvested from the wild state. Columbus found corn being cultivated on Haiti, where it was called "Mahiz". From this Arawak Indian word was derived the name maize that is used in Europe to distinguish the cereal from other grains which are called "Corn" (Hunt, 1915).

With the discovery of the New World, maize cultivation was learnt from these Indian tribes. Christopher Columbus was probably the first to see and describe this new cereal, found by members of his crew in Cuba and he named it maize. It is most likely that Columbus or some of his contemporaries were the first to introduce maize to Europe.

Using Vavilov's concept of centre of origin, the high lands of Peru, Bolina and Ecuador would appear to have the best claim as the starting point of maize on account of the great diversity of the native forms in that region (Jugenheimer, 1958). On the other hand, maize may have originated in Mexico or Central America because this area is considered to be the home of teosinte grass (*Euchlaena mexicana; Schard.*), a near relative of maize, which shows a wide range of types in this area.

The introduction of maize in the pre Columbian era would have taken place through the usual land trade routes in operation between the east and west.

Globally, maize is grown on 140 million hectares with an annual production of about 600 million tonnes. Tropical maize is grown in 66 countries and is of major economic significance in 61 of these countries each having 50,000 ha with an annual production of 111 million tonnes. The average yield of maize in the tropics is 1.8 tonnes/ha as against the global average of 4.2 tonnes/ha. The average yield of temperate maize is 7 tonnes/ha (International Maize and Wheat Improvement Center [CIMMYT], 1994).

In India, maize is grown in almost all the states. It is fourth in area (6.59 million ha), next to rice, wheat and sorghum, but third in production. The productivity during the year 2001-2002 was 2018 kg/ha as against 1841 kg/ha of the previous year. The marginal increase

in area has been reported from the state of Gujarat, Bihar, Andhra Pradesh, Madhya Pradesh, Rajasthan and Uttar Pradesh.

Maize is relatively a warm weather crop and requires 120-140 days to mature. The maize crop is so important that some or the other part of the plant is utilized either for food, fodder or industry. The grain is used either for human consumption or as a feed for poultry and cattle, the green leaves; stem and cob shelling are used as fodder for cattle. The grain is also used to produce corn oil, syrups, starch, beverages, vitamins, amino acid, alcohols, etc. In India, the use pattern of maize is 65% for human consumption, 15% for livestock including poultry, 18% for industry use and 2% for seed purposes.

Further, the demand of maize for human consumption, livestock and poultry and for industrial use is likely to increase to about 20 million tonnes by the year 2008 (Kamble *et al.*, 2004). To meet the above requirement, the existing productivity level of 1.6 tonnes/ha will have to be doubled. One of the important constraints identified for low level of productivity is the lack of mechanization for the cultivation of maize crop. Although the equipments for different operations have been developed but their adoption at the farmers level have been poor.

Maize belongs Poaceae family of Angiosperms. It is a robust erect annual grass. Some varieties may reach 15 feet or more in height, while others rarely exceed 1½ feet at maturity. It has fibrous root system, broad leaves arranged in two alternate vertical ranks, split leaf sheaths, cylindrical stems with solid nodes and flowers in more or less chaffy spike lets (Fig. 1).

The Inflorescences are monoecious, flowers on staminate tassel are borne in many spike like racemes, which together form large spreading panicles, which terminate the stems. A pistil late inflorescent is borne on one or more spikes of the leaves. There are eight or more rows of spikelets on a thickened axis (cob) and the whorl is enclosed in felicitous bracts (husks). Protruding from the tops of these bracts are the styles called silks. The styles are long and slender and may be fertilized throughout their length.

Maize requires fertile, well-drained clay loam soils with high humid content. The soil pH should be almost neutral (6.5-7.0) under good management and fertilization, it can be grown in sandy loam to loam soils.

Maize is normally planted at a depth of 5 to 8 cm, provided moisture is adequate at that depth. It absorbs water and begins to swell. This proceeds at a faster rate at higher temperatures, which are prevalent in most tropical environments in the summer season. Under these conditions, the seed starts to germinate in two to

Fig.1: Mature maize crop.

three days. In the winter season and in other low soil temperature conditions such as highlands, the process is delayed and radical emergence may take as many as six to eight days (Onderdonk and Ketcheson, 1972).

The crop is very much popular among the farmers due to its dual utility as grains and as a fodder. The green portion of the plant left after removing cobs is used as a forage source.

The maize plant is an excellent fodder for both milch cattle and draft cattle. It is used as fodder at various stages of plant growth, particularly from tasselling onward. The maize when grown for forage is most nutritious at silk stage. As per an estimate maize contributes approximately 240 million t GFY in India (Annonymus, 1987). It does not have problems of prussic acid or hydrocyanic acid and therefore may be used as fodder even before flowering or in dry weather. Maize with ears at the dough stage of grain development is best for use as green fodder and silage preparation. It surpasses all other fodder crops, in dry matter production and digestible nutrients per hectare. Ensiled maize for fodder is more commonly used in temperate environments where cold weather limits maize growth and season length. It is not common in tropical countries where maize is grown in more than one season. Stover left after the harvest of the grain is also used as fodder,

particularly if stay green type varieties are used where stalks and leaves are still green at harvest time.

The Bundelkhand region (Fig. 2a, b) has a huge livestock population solely dependent upon the grazing resources. At present the grazing area has drastically squeezed due to its allotment to landless persons and the remaining land is heavily degraded due to overgrazing. Thus, the cultivated crops become the major source of dry and green fodder supply to the livestock. Hence, it would be pertinent to highlight some aspects relating to fodder crops grown in the region.

In Bundelkhand 1.61% of area of cereal crops is covered under maize, which amounts to 0.35 lakh ha (Table 1). The area under maize at district level in the region reveals that Southern Bundelkhand is the main maize growing area. The highest acreage under this crop has been recorded in Lalitpur (16.91 lakh ha.) followed by Sagar, Jhansi, Panna, Tikamgarh and Damoh districts where the area under maize ranges from 0.02 to 0.04 lakh ha. The districts of northern plain, which receive less rainfall, recorded insignificant area under maize crop in comparison to Jowar as maize requires 5-6 irrigations. The production of maize is almost nil in the districts of Banda, Jalaun and Hamirpur as very meager area was recorded under this crop. The highest production (0.32 lakh tonnes) is obtained in Lalitpur followed by Jhansi district (0.07 lakh tonnes). The

Fig.2: (A) Location of Bundelkhand in India

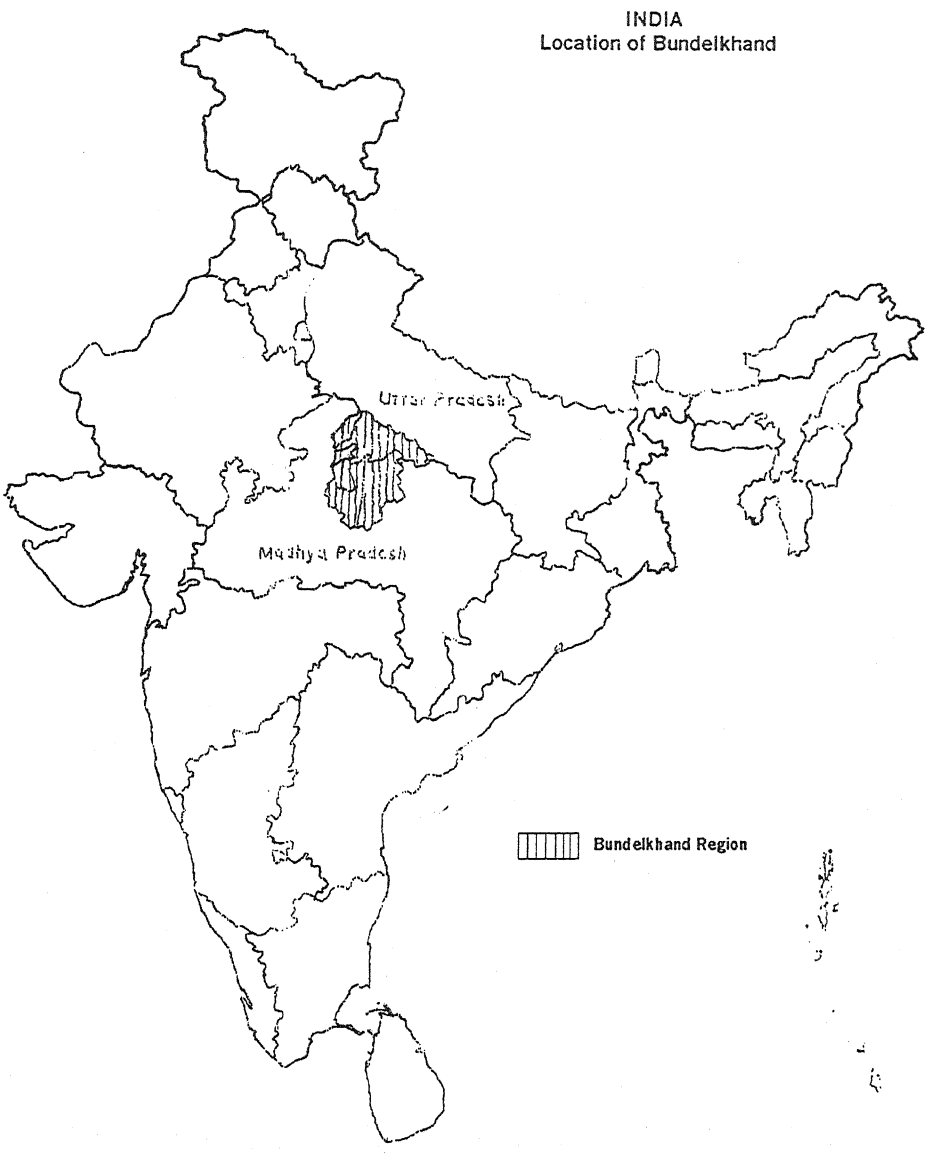


Fig: 2A location of Bundelkhand in India.

Fig.2: (B) Different districts of Bundelkhand region.

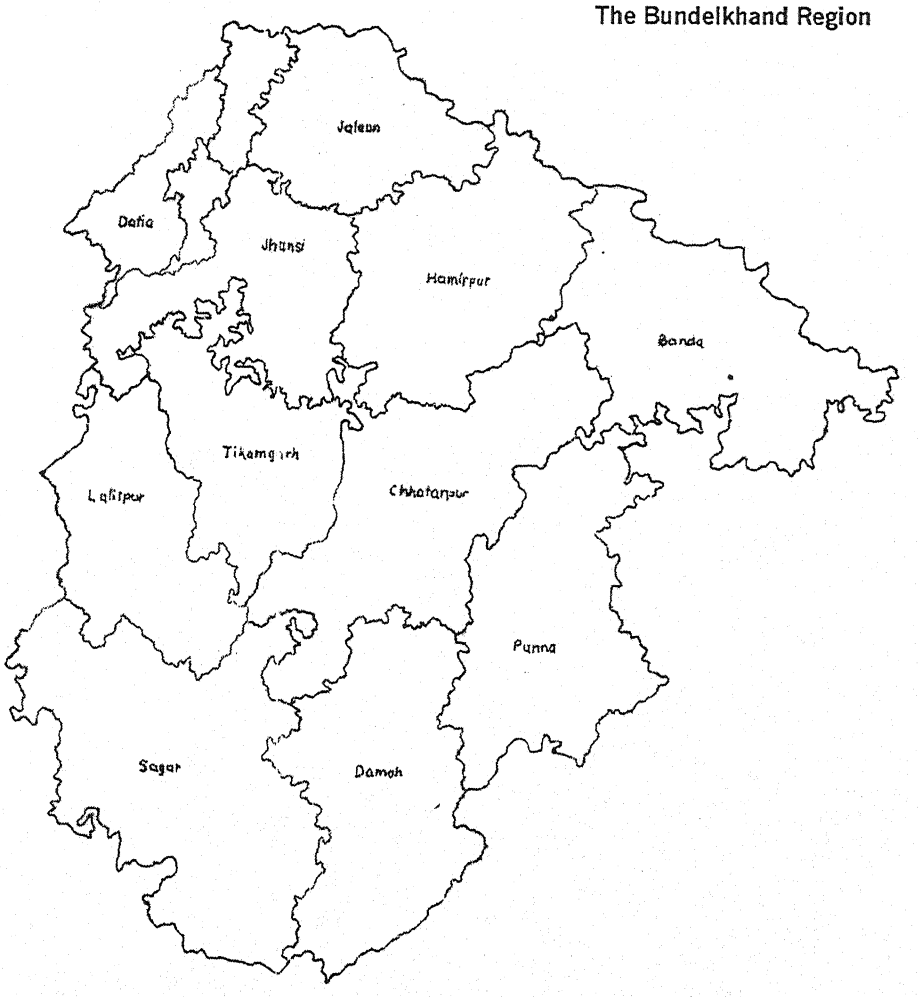


Fig. 2B: The Bundelkhand Region

Table 1: Area under cereals in different districts of Bundelkhand*

('000ha)

District	Paddy	Wheat	Jowar	Maize	Bajra	Barley	Other cereals	Total Areas
Banda	81.47	173.18	80.23	0.01	14.00	11.99	3.78	364.66
Jalaun	4.27	79.91	28.46	0.02	17.16	9.15	0.03	139.00
Hamirpur	5.10	151.09	100.87	0.01	1.14	4.88	3.10	266.19
Jhansi	4.14	101.32	54.24	3.98	0.02	2.75	0.64	167.09
Lalitpur	13.00	72.77	36.62	16.91	-	2.98	7.19	149.47
Datia	1.20	39.60	19.30	1.50	0.80	1.00	0.10	63.50
Tikamgarh	30.80	87.60	36.10	2.50	-	7.60	19.00	183.60
Chhatarpur	21.10	112.90	29.80	0.90	0.10	19.20	3.70	187.70
Panna	49.10	79.40	8.60	3.20	-	5.40	-	145.70
Damoh	51.20	97.80	18.20	2.10	0.10	0.10	0.20	169.70
Sagar	15.20	263.60	21.70	4.40	-	0.80	1.70	307.40
Region	276.58	1256.17	434.12	35.53	33.32	65.85	39.44	2141.01

* Grassland and Fodder Atlas of Bundelkhand (1997)

production in the southern districts ranges from 0.01-lakh t in Chhatarpur to 0.06 lakh tonnes in Sagar district. The average yield of maize in the region has been worked out as 1.51 tonnes/ha. It ranges from 0.85 tonnes/ha in Panna to 2.00 tonnes/ha in Hamirpur. A high yield was obtained in all other districts of the U.P. part of Bundelkhand i.e. 1.90 tonnes/ha. In maize growing districts of southern Bundelkhand the yield was recorded below regional average. Hence, it can be raised in these districts (Table 2 & 3).

Besides meager area under fodder cultivation pests disease and weeds are also limiting factors for fodder availability about 34.8 percent losses are being incurred by these pests, diseases and inflicted weeds (Cremer, 1967) (Table 4).

Of the sixty-one diseases of maize recorded so far, fifteen fungal and one nematode problem are considered as major constraints in maize production. The major diseases are four foliar diseases; two pre-flowering and three-post flowering stalk rots, four downy mildews and two sheath blight diseases (Renfro & Ullstrup, 1976). In Bundelkhand region, maydis leaf blight, turcicum leaf blight, bacterial spots and rust are most frequently accrued. The losses caused by various diseases in maize are presented in Table 5.

Maydis leaf blight or southern leaf blight (SLB) {(*Helminthosporium maydis* Nisik. [Syn. *Bipolaris maydis*

Table 2: Production under cereals in various districts of Bundelkhand*

('000 Tonnes)

District	Paddy	Wheat	Jowar	Maize	Bajra	Barley	Other cereals	Total
Banda	42.12	191.00	47.20	-	8.42	12.70	2.40	303.84
Jalaun	2.30	123.70	21.40	-	10.30	13.75	-	171.45
Hamirpur	2.70	178.90	56.50	-	0.70	4.50	2.00	245.30
Jhansi	2.20	150.60	44.70	7.60	-	2.51	0.30	207.91
Lalitpur	8.00	92.20	27.40	32.30	-	2.74	4.13	166.77
Datia	0.60	70.10	17.90	1.90	0.90	1.40	-	92.80
Tikamgarh	14.50	150.60	37.60	2.60	-	5.10	9.00	219.40
Chhatarpur	14.80	156.80	27.50	1.00	-	22.00	9.30	231.40
Panna	11.90	57.60	6.00	2.70	-	3.80	1.50	83.50
Damoh	24.60	77.10	18.50	3.10	0.10	0.10	1.12	124.62
Sagar	8.00	180.50	19.80	5.90	-	0.60	1.40	216.20
Region	131.72	1429.10	324.50	57.10	20.42	69.20	31.15	2063.19

* Grassland and Fodder Atlas of Bundelkhand (1997)

Table 3: Average yield under cereals in various districts Of Bundelkhand

(kg/ha)

District	Paddy	Wheat	Jower (*Kharif*)	Maize	Bajra	Barley
Banda	517	1102	568	1900	682	1057
Jalaun	529	1376	753	1900	602	1503
Hamirpur	529	1252	560	2000	602	1007
Jhansi	529	1486	825	1900	602	921
Lalitpur	620	1267	748	1900	-	921
Datia	601	1843	932	1209	734	1422
Tikamgarh	496	1909	1042	1050	-	669
Chhatarpur	740	1355	920	1088	323	1146
Panna	255	755	702	849	294	712
Damoh	502	820	1016	1465	1057	1074
Sagar	551	714	912	1342	615	680

Table 4: World Crop losses Caused by Various Pests and diseases (in %).

Commodity	*Insect Pests*	Diseases	Weeds	Total
Wheat	5.0	9.1	9.8	23.8
Rice	26.7	8.9	10.8	46.4
Maize	12.4	9.4	13.0	34.3
Other Cereals	6.6	8.6	12.1	27.3
Potatoes	6.5	21.8	4.0	32.3
Succulents & sugar beets	16.6	16.5	12.2	45.3
Vegetables	8.7	10.1	8.9	27.7
Fruit crops	5.8	16.4	5.8	28.8
Oil crops	11.5	10.2	10.8	32.5
Stimulant's	11.4	14.9	10.5	36.6
Fiber crops	14.4	11.8	6.3	32.3

Table 5: World Maize (Region wise) Annual losses Caused by Various Pests and diseases (in %).

Regions	***Insect Pests***	***Diseases***	***Weeds***	**Total**
N & C America	11.7	9.4	7.8	28.9
S. America	20.0	10.0	10.1	40.0
Europe	5.0	3.0	6.0	14.0
Africa	20.0	13.6	35.0	68.6
Asia	10.0	12.0	15.0	37.0
Oceania	5.0	5.0	7.0	17 0
USSR & China	12.4	9.4	13.0	34.6

(Cramer, 1967)

(Nisik.) Shoemaker, *Drechslera maydis* (Nisikado) Subram. & Jain] Perfect state *Cochliobolus heterostrophus* (Drechs.) Drechsl.)} occupies the second place next to turcicum blight (Table 6).

D. maydis (*H. maydis*) has been reported to cause a number of serious diseases in graminaceous plants. Southern corn blight caused by *D. maydis* was reported as a serious epidemic and threatened the corn growers in tropical region especially after the release of male sterile clones. *D. maydis* has also been reported on maize causing a leaf spot disease in India and other graminaceous hosts (Table 7& Fig. 3). However, this seems to be the first report of *D. maydis* on *C. speciosus*. (Gupta, *et al,* 1978).

Lesions on SLB infected leaves are tan, spindle or elliptical with yellow green or chlorotic halos. Later lesions often develop dark, reddish brown borders and may occur on leaves, stalks and husks. A black, mold may cover kernels in the infected ear.

The mycelium is dark grey. The light green to brown conidiophores, which arise in groups of two or three on the dead leaf spots, produce cigar-shaped, smokey grey conidia.

The fungus over winters as dormant mycelium and spores in maize debris were bound in the field and on kernels in cribs, bins and elevators. Primary infections result from conidia carried by wind or splashing water to

Table 6: Yield loss by diseases under experimental conditions in susceptible cultivars in India.

S. N.	Diseases	Loss (%)	Reference
1	Bacterial stalk rot	100	Thind and Payak, (1970)
2	Pythium stalk rot	100	Payak and Sharma, (1978)
3	Late wilt	50.9	Payak and Sharma, (1978)
4	Charcoal rot	39.5	Payak and Sharma, (1978)
5	Maydis leaf blight	30.3	Payak and Sharma, (1978)
6	Turcicum leaf blight	66	Payak and Sharma, (1978)
7	Common rust	32	Sharma et al., (1982)
8	Brown spot	27	Lal and Chakravarti, (1976)
9	Brown stripe downy mildew	63	Anonymous, (1972)
10	Bended leaf and sheath blight	40.5	Singh and Sharma,(1976)

Table 7: Host Range of *Drechslera* spp. in India

S. N	Species	Host	Host Family	Locality
1.	*D. australiensis*	*Air* *Arachis hypogaea L.* *Capsicum annuum L.* *Cynodon dactylon (L)* *Dracuena americana L* *Oryza sativa L* *Polygonum sp.* *Solanum melongena L.* *Sorghum halepense L.*	- Leguminaceae Solanaceae Gramineae Liliaceae Gramineae Polygonaceae Solanaceae Gramineae	Tiruchirapalli (T.N.) Jaipur (Raj) Galior (M.P.), Jalna (M.S.) New Delhi New Delhi New Delhi Dibrugarh (Assam), Gangtok (Sikkim), New Delhi
2.	*D. anvenae*	*Avena stiva L.*	Gramineae	Sibpur, (W.B.); Bihar, New Delhi, Gorakhpur, Kanpur (U.P.)
3.	*D. euphorbiae*	*Euphorbia helioscopia L* *Euphorbia geniculata L*	Euphorbiaceae Euphorbiaceae	New Delhi Jabalpur, (M.P.)
4.	*D. graminea*	*Capsicum anuum L* *Digitaria sanguinalis L* *Sordeum vulgare L*	Solanaceae Gramineae Gramineae	New Delhi Gauhati, (Assam) Agra, Deharadun, Kanpur, Orai, (U.P.), Ajmer, Marwar, Jaipur (Raj), Pusa, Dhdi,

		Paspalum Conjugatum	Gramineae	Motihari, Sabour, (Bihar), New Delhi, Diphu, (Assam) Dibrugarh, (Assam)
5.	*D. hawaiiensis*	*Buddleia asiatica L.* *Cajanus cajan L.* *Capsicum annuccm L.* *Chloris gayana* *Common weeds* *Cycas circinalis L* *Moringa oleifera* *Rottboellia exaltata L.* *Sees of orgza sativa L.* *Sorghum vulgare* *Triticale* *Triticum aestivum L* *Zea mays L.*	Buddleiaceae Pepilionaceae Solanaceae Gramineae - Cycadaceae Moringaceae Gramineal Gramineal Gramineal Gramineal Gramineal Gramineal	Khanapara, (Assam) Pune, (M.S.) Gwalior, (M.P.) Dharwad, (Karnataka) Dharwad, (Karnataka) Bhubaneshwar (Orissa) Rahuri (M.S.) Tattapani (Meghalaya) Gurdaspur (Punjab) Dharwad (Karnataka) Dharwad (Karnataka) Hyderabad (A.P.), Dharwad, (Karnataka) Dharwad (Karnataka)
6.	*D. heveae*	*Hevae brasiliensis*	Euphorbiaceae	Port Bliar (Andaman and Nicobar Islands)
7.	*D. maydis*	*Zea mays L.*	Gramineae	Diphu (Assam), New Delhi, Ghaziabad, Almora (U.P.), Warangal (A.P.), Shillong (Meghalaya), Kalyani (W.B.), Kdhapur (M.S.), Mandor (Raj.), Sankeshwar

				(Karnataka)
8.	*D. nodulosa*	*Eleusine coracana L.*	Gramineae	Chotta Nagpur, Sabour, (Bihar), Coimbatore, Vayitri, Wynaad (T.N.) Almora, Kumaon, (Utranchal)
9.	*D. oryzae*	*Oryza officinalis* *Oryza sativa L.*	Gramineae Gramineae	Bhubaneshwar (Orissa) Malda (W.B.), Ratnagiri (M.S.), Cuttack (Orrissa), Idukki, Kottarakara (Kerala), Pusa, Darbhaga (Bihar), Jammu (J&K), Shillong (Meghalaya), Hyderabad (A.P.), Virudhunagar, Coimbatore, Madras (T.N.), Gurdaspur Karparthala (Punjab)
10.	*D. ravenelii*	*Eragrostis sp.* *E.plumose* *Pennisetum typhoides* *Sporobolu spp.*	Gramineae Gramineae Gramineae Gramineae	Majhgawan (U.P.) Varanasi (U.P.) New Delhi Imphal (Manipur), Bhowali (U.P.)
11.	*D. sorokiniana*	*Agropyron droborii* *Apluda mutica L.* *Aristida fumiculata L* *Avena sativa L* *Capsicum annuum L.*	Gramineae Gramineae Gramineae Gramineae Solanaceae	Wellington (T.N.) Bhind (M.P.) Bhind (M.P.) Faridkot, Kapurthala, Hoshiarpur (Punjab), Nainital (Utranchal)

		Dioscorea composita	Dioscoreaceae	Valpoi (Goa)
		Hordeum vulgare	Gramineae	New Delhi
		Oryza sativa L	Gramineae	Ludhiana, Gurdaspur, Ropar, Patiala, Amritsar, Ferozpur (Punjab), Kulu (Himachal Pradesh) Impal (Manipur)
		Paspalum notatum	Gramineae	Dharwad (Karnataka)
		Triticum aestivum	Gramineae	Junagadh (Gujrat), Jorhat (Assam), Bihar; New Delhi; Imphal (Manipur)
		Zea mays L	Gramineae	Faribkot, Kapurthala, Hoshiarpur (Punjab)
12	*D. splcifera*	*Capsicum annuum*	Solaneceae	Junagadh (Gujrat)
		Coccinia indica	Cucurbitaceae	Warangal (A.P.)
		Musa paradisiacal	Musaceae	Bhagalpur (Bihar)
		Populusnigra	Salicaceae	Ludhiana (Panjab)
		Soil	-	New Delhi
		Titicum aestivum	Gramineae	New Delhi, Hyderabad (A.P.)

Fig.3: Occurrence of *Drechlera spp* in India.

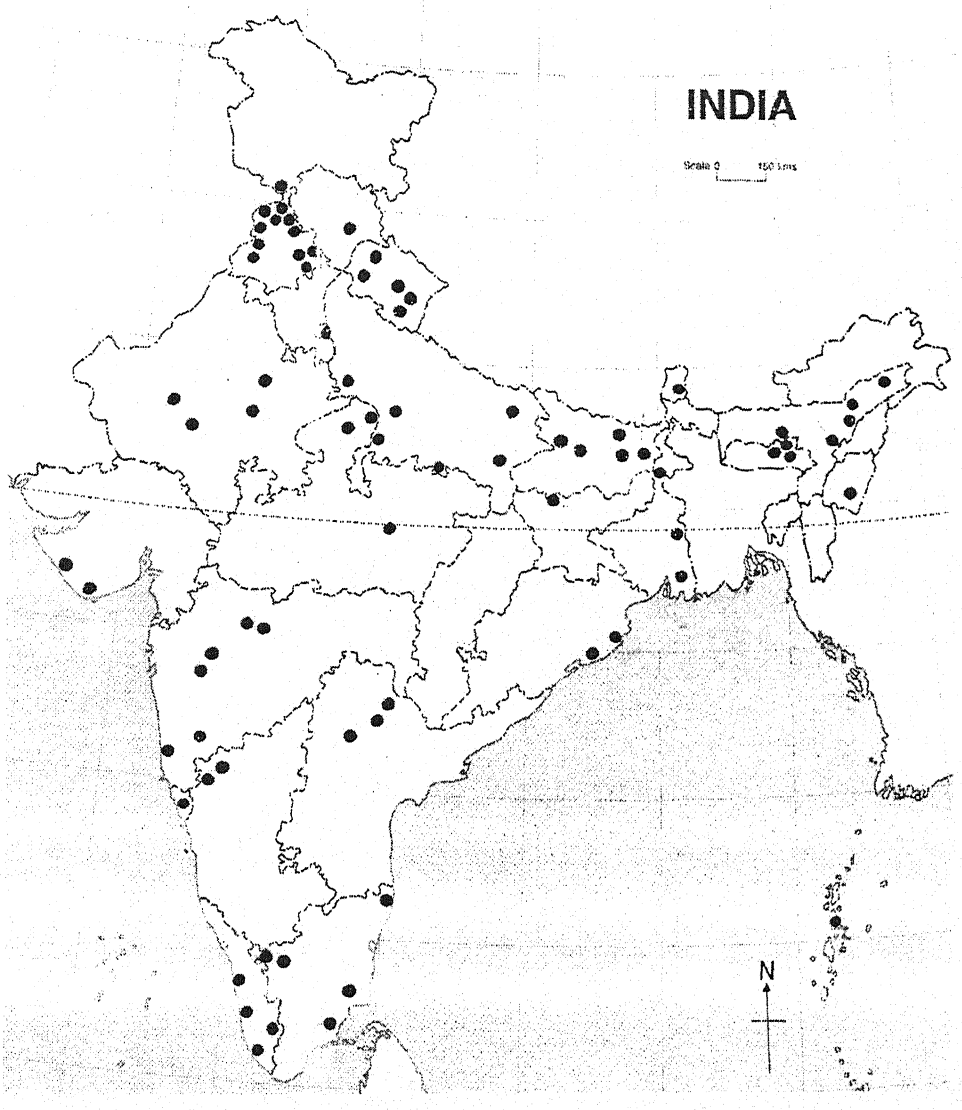


Fig.3: Occurrence of *Drechslera* spp. In India

growing plants. Sporulations on lesions cause secondary infection.

Realizing the importance of the crop in the area and losses caused by the pathogens, the present studies were formulated with following objectives:

1. Prevalence and severity of disease in the area.
2. Symptomatology of the disease under natural field conditions.
3. Morphological & cultural characteristics of the pathogen.
4. Mode of perpetuation and optimum growth requirement.
5. Source of resistance, assessment of qualitative and quantitative attributes of patho-system and their categorization.

REVIEW OF LITERATURE

Chapter-2

REVIEW OF LITERATURE

In India, the success of agriculture, particularly of the rainy season (Kharif) crops like maize, sorghum, depends primarily on the monsoon. Generally, maize is planted at the onset of monsoon and harvested in October. High relative humidity and high temperature is congenial for the development pathogens. Maydis leaf blight or Southern crop leaf blight (SLB) caused by *Drechslera maydis* (Syn. *Bipolaris maydis* or *Helminthosporium maydis*) is the perfect state *Cochliobolus heterostrophus* occupies fairly high place.

According to Kaiser and Das (1989) Race O of *Cochliobolus heterostrophus* predominates on maize in India while race 'T have been detected occasionally. The race T was first appeared in 1960. The occurrence of race

T is reported for the first time in India and it is believed that it may have transmitted through seeds of the exotic cv. CIMMYT bulk-5 imported from the USA.

In Assam and Meghalaya states of India *D. maydis* (*Cochliobolus heterostrophus*), was collected on maize by Vinh and Sarbhoy (1991).

The isolates obtained from maize, infected with southern leaf blight in Japan were assigned to *B. maydis* (*Cochliobolus heterostrophus*) on the basis of morphology and cross-fertility. All were equally virulent to lines with T-cms, C-cms and N type cytoplasm. None of the culture filtrates of the isolates caused leaf wilting in lines with *T-cms* and *C-cms* and no host-specific toxin was detected. All the Japanese isolates were therefore, assigned to race O. Four complements of aggressiveness (infection efficiency, lesion area, sporulating lesion ratio and sporulation) of 39 isolates of race O were analyzed using single spore inoculation method in maize seedlings of the susceptible inbred line Pa91. Infection efficiency and sporulation differed among the isolates but no difference was detected in the ability of lesions to enlarge. There was no variation among the regions from which the isolates were collected. It was conducted that the Japanese isolates could not be grouped by principal component analysis of aggressiveness (Tsukiboshi *et al.*, 1995).

Tsukiboshi *et al.*, (1996) also reported *B. maydis* (*Cochliobolus heterostrophus*) from 35 sites in Japan. Their matting types were determined by pouring with tested isolates, as in the case of four isolates obtained from *Panicum spp.* and *Bothriochloa* spp. All isolates were equally pathogenic to the differential lines of maize with *T-cms*, *C-cms* and *N-C* type cytoplasms in spore-spraying tests. All the isolates were assigned to race O. The 75 isolates from maize produced only small and chlorotic lesions in the *rhm* resistant maize line, 1 isolate caused severe symptom with typical and large lesions. The increased ability to cause lesion enlargement of the isolate in the *rhm* resistant line was attributed to the increase in the number of appressoria formed at the time of spore germination and faster hyphal extension in the host cells. This is the first report on a *C. heterostrophus* isolate virulent to the *rhm* maize line in the world.

Harlapur *et al.*, (2000) reported recent information on maize diseases in north Karnataka (India). *Turcicum* leaf blight [*Exserohilum turcicum* (*Setosphaeria turcica*)] was the major disease (53.5% incidence) affecting maize, particularly during the Kharif season. Charcoal stalk rot (*Macrophomina phaseolina*) appeared in major proportions during the Rabi season (16.5 incidence). The incidence of other foliar diseases [*Maydis* leaf blight {*Drechslera maydis* (*Cochliobolus heterostrophus*)}], Polysora rust (*Puccinia polysora*) and common rust

(*Puccinia sorghi*) was moderate, Downy mildew (*Peronosclerospora sorghi*), brown spot (*Physoderma maydis*) and *Phaeosphaeria* leaf spot (*Phaeosphaeria maydis*) incidence were observed in traces. During Rabi season, charcoal stalk rot and *Fusarium* stalk rot (*Fusarium moniliforme* {*Gibberella fujikuroi*}) incidence found to be moderate to severe.

Drechslera maydis (Nisik) Subram and Jain, *Bipolaris maydis* (Nisik.) Shoemaker, *Helminthosporium maydis* Nisik. Syn.), teleomorph *Cochliobolus heterostrophus* (Drechs) occurs as races O, T and C. Race O was found to have a higher saprophytic ability than race T; only about 4% of the recovered spores were race T (Blanco and Nelson, 1972). Race C was reported in China infecting maize with C-cytoplasm (Wei *et al.* 1988). Race T is characterized as specific for certain cytoplasms such as the widely used T (Texas) type for male sterility. The P-cytoplasm from south America and a few other cytoplasms are also known to be susceptible (Hooker *et al.*, 1970); is a weak parasite on resistant plants in the field; seedlings are more easily infected; produces a pathotoxin; attacks the leaf, leaf sheath, husk, shank, ear and stalk tissue of the plant; reproduces rapidly in susceptible plants and; may have a lower temperature optimum than race O; race O shows little or no specificity to plant cytoplasms; produces only limited amounts of a non specific phytotoxin; infects

leaves mainly, producing smaller lesions with parallel sides and little chlorosis; seems to reproduce less rapidly than race T on susceptible plants; tends to be limited by temperature and climate to the warmer part of the US (Hooker *et al.*, 1970). Spore production is influenced by temperature in both races with race T being most sensitive. More lesions formed at 30° C than at 15 or 22.5°C (Warren, 1975). Lesion size increases in almost a straight-line relationship with increasing dew periods and colonization temperatures (Nelson and Tung 1973). In the 1970's *B. maydis* induced southern maize blight epidemic causing loss in maize [with cytoplasmic male sterility gene (CMS-T)] of more than 1 Billion US Dollar (Ullstrap, 1972).

Foroutan and Rahimian (1990) reported the occurrence of *Drechslera maydis* [*Cochliobolus heterostrophus*] in maize fields in Sari during the summer, constitutes a new record for Iran. *Helminthosporium turcicum* [*Setosphaeria turcica*] was also isolated from some infected leaf samples.

2.1 Plant disease assessment

The lack of reliable data to define the importance of disease in world agriculture may well have retarded the progress of plant pathology as much as any other single factor. Losses due to disease are substantially higher in developing countries and unfortunately more severe in

countries that can least afford them. Assessment of disease presents the initial data critical in plant protection programs. In order to have and sustain sound planning or management of plant pathology investment, we need estimates of crop losses. Priorities in resource allocations must be established during planning stages (James, 1968). There has been no standard protocol for disease assessment, although many forums have been devised to look for practical, efficient and accurate assessment of disease intensity. A globally accepted standard method would encourage data comparison, improvement of communication and interpretations of results between professionals and ultimately to the consumer, the farmer (Wastson, *et al.*, 1990). Despite the great need for a standard, there has not been a universally accepted formula for disease measurement. A commonly used system that of Horsfall and Barratt (1945) suggests that according to Weber-Fechner law, the eye distinguishes according to the logarithm of the light intensity. Therefore, the grading was to be used on equal ability to distinguish, but not on equal disease. Below 50% it sees the amount of disease free tissue. They proposed a 12-category scheme of disease assessment. However, Hebert (1982) indicates that the underlying assumption of the Horsfall-Barratt system is false and that we are devoid of data supporting one scale over the other or even the superiority of a scale over direct visual disease assessment. Slopeck (1989) stated

that the Horsfall and Barratt system is more rapid that direct visual estimates because one need only determine severity of disease is within the percent leaf area diseased (PLAD) range boundaries of a rating category. Slopeck (1989) used a 1-5 scale containing ranges from 0 to 100 as per cent leaf area diseased categories, but also noted that visual assessments in some situations may be most valuable.

Field disease measurement in varietals trial and measurements production can help estimate reductions in yield and quality (Large, 1965). Duveiller (1994) developed pictorial disease assessment keys for bacterial leaf streak determined by measuring diseased leaves in cereals from 1 to 75% diseased area. In Uganda Adipala *et al.*, (1993a) used 0 to 75% severity scale for maize northern leaf blight and observed 20 plants along the geographical cardinal points. On selected farms, Adipala *et al.*, (1993a) examined five plants to the north, south, east and west resulting in a sample of 20 plants per site and then visually assessed disease severity. Disease severity or the extent of tissue coverage by lesions rating on a scale of 0 to 5 was used by Nicholson and Warren (1975), O'Brien and Bruggen (1992) used ten participants and three severity scales to test accuracy and precision for corky root of lettuce. One of the scales which they called mature plant scale had seven levels from 0 to 6, the other scale known as seedling scale had

ten levels from 0 to 9 and third one was the H-B scale which had 12 levels, 1=0%, 2=0-3%, 3=3-6%, 4=6-12%, 5=12-25%, 6=25-50%, 7=50-75%, 8= 75-87%, 9=87-94%, 10=94-97%, 11=97-100%, 12= 100%. They found that no scale was better than the other in each situation but the scale for mature plants was more precise and accurate. Saghaai Maroof *et al.*,(1993) used seven inbred to evaluate grey leaf spot in Virginia. They used two methods namely; disease index and disease severity. The disease index was a scale of 1-5 where 1= no symptoms; 2= moderate lesion development below the leaf subtending the ear; 3= heavy lesion development on and below the leaf subtending the ear and a few lesion above it; 4= severe lesion development on all but the upper most leaves, which may have a few lesions; and 5= all leaves dead. They expressed disease severity as diseased leaf area divided by total leaf area multiplied by 100 assessed on the ear-1 leaf and all leaves above it. This study found that the disease index is a better method for general resistance screening of germplasm with multiple rating. Gaunt (1995) proposes that remote sensing, imaging and positioning hardware and software have provided new technologies for assessing disease severity and for sampling. Nutter and Schultz (1995) reported that use of computerized disease assessment training programs such as Disease Pro, can greatly enhance accuracy and precision of visual disease assessments. Maize crop for assessment is found in the field in many

stages of growth as illustrated by Hanway (1963). Assessment depends on the pathogen involved, the crop that is diseased and its growth stage. It is critical that the time of survey to coincide with the pathogen peak activity and this time the host shows most evidence of attack.

The Food and Agricultural Organization (FAO) of the United Nations has developed publications offering guidelines on disease assessment method (Botrel, 1979). Despite the need for a standard, there has not been a universally accepted formula for disease measurement. Disease assessment generates a large data base which is expensive to collect and therefore, should be fully interpreted and feed back given to the farming community (James, 1968).

Elliot and Jenkins (1946) assessed *H. turcicum* severity on a 0 to 5 scale as follows; 0.5=very slight infection, one or two restricted lesions on the lower leaves, 1=slight infection, a few scattered lesions on the lower leaves, 2= light infection, moderate number of lesions on lower leaves, 3= moderate infection, abundant lesions on lower leaves and few on middle leaves, 4= heavy infection, lesions abundant on all leaves, and extending to upper leaves, 5= very heavy infection, lesions abundant on all leaves plants may be prematurely killed.

Pataky (1992), in a study of resistant and susceptible sweet corn used a rating scale of 2% to 90% generated from a computer program. DISTRAN developed by Tommerlin and Howell (1988). The evaluation was based on the primary ear leaf and top and bottom leaves which accounted for 33 to 40% of the total leaf area in sweet corn. Susceptible corn consistently showed yield losses unlike those with *Ht* genes of resistance. Pedersen *et al.,* (1986) using a 1-9 scale adopted from Perkins and Hooker (1981), evaluated levels of resistance to *E. turcicum* by inoculating inbred lines B37, B37HtZ and oh43Ht3 in the green house and in plots. They found some lines classified resistant in the green house were susceptible in different locations, probably due to temperature, relative humidity, and light variations. Manwiller *et al.,* (1985) rated maize at Muguga for reactions to rust and Northern maize blight but did not challenge the lines. Darrah and Mukuru (1977-80) evaluated maize crosses for hybrid production for rust and northern leaf blight incidence on a 0-5 scale where 0-represented no symptoms and 5 very severely attacked based on natural field infection, without artificially inoculating the progenies. During this period rust scores had a mean value of 3.07 while blight was even lower at 1.93. Scores of 3.8 and 3.3 were recorded for rust and northern leaf blight, respectively, (Darrah 1974, Darrah *et al.,* 1975, 1976).

2.2 The Pathogen Taxonomy

Helminthosporium Link Fr. is a generic name well known to plant pathologists. It has been applied to some important pathogens, such as *H. maydis* Nisik & Miyake, *H. Oryzae* Brede de Haan, *H. turcicum* Pass and *H. teres* Sacc. These and similar species are commonly associated with leaf spots or blights, foot rots and other disease syndromes on cultivated and wild *Poaceae*. Their ability to cause devastating disease has occasionally resulted in famine and loss of human life (Padmanabhan, 1973) or in great economic loss (Ullstrup, 1972).

The genus *Helminthosporium* was established in 1809. Modern accounts of the type species, *H. velutinum* Link, S.F. Gray, have been given by Luttrell, (1963), Ellis (1971) and Hughes (1980). Conidiophores arise from a rudimentary to conspicuous stroma up to 400-µm heights and 700 µm wide. They occur singly or in tufts and are cylindrical to slightly tapered, dark brown, unbranched, straight or flexuous, septet and produce conidia through small pores in the walls of the distal and intercalary cells. Conidia are formed laterally, often in verticals below septa, while the conidiophores are elongated. Production of terminal conidia usually signals cessations of conidiophores growth, although mature conidiophores may resume apical growth if transferred into a moist atmosphere (Luttrell, 1963). Conidia are obelavate to obclavate rostrato, straight or curved, sub

hyaline to brown, transversely disto-septate, smooth and rounded to truncate at the base, which bears a conspicuously darkened hilum or "scar".

The genus *Helminthosporium* became the repository for a large number of newly described taxa, only some of which were congeneric with the type species. Subsequently many have been assigned to genera such as *Alternaria, Cercosporidium, Corynespora, Deightaniella, Embellisia, Pseudocercospora, Spiropes* and *Stigmina*: other have been shown to be synonyms of species in *Cladosporium, Mycovellosiella, Pleurophragmium, Teratosperma* and other genera.

(i) Segreate and Synonymous Genera

The *Helminthosporium* species from grasses were segregated by Nisikado, (1928, 1929) into two subgenera, *Cylindro-Helminthosporium* and *Eu-Helminthosporium* species with straight cylindrical conidia that germinate by one or more germ tubes from any cell were grouped in the former subgenus, while species with fusiform, often curved conidia germinating only from end cells were placed in *Eu-Helminthosporium.*

(a) Drechslera: *Drechslera* was established by Ito (1930) to accommodate fungi previously assigned to the subgenus *Cylindro-Helminthosporium.* Members of the new genus were characterized, as having "Cylindrical", not curved conidia, germinating from every cell and

associated with *Pyrenophora. Drechslera tritici-vulgaris* (Nisik) Ito (=*D. tritici-repentis* [Died.] Shoem.) was designated as the lectotype by Hughes (1958).

(b) Bipolaris: Shoemaker, (1959) established the genus *Bipolaris* for species that Nisikado, (1928) had placed in his subgenus *Eu-Helminthosporium.* Conidia were described as fusoid, straight or curved, and germinating by one germ tube from each end. Taxa with a protuberant conidial hilum, such as *B. rostrata (Drechsler) Shoem* and *B. turcica* (Pass.) Shoem, were included in the new genus. These species had been shown by Luttrell, (1957) to have ascigerous states in *Metosphaeria* (later as *Trichometasphaeria*); while other species of *Bipolaris* had nonprotruding hila and the teleomorphs of some were in *Cochliobolus.*

(c) Exserohilium: Leonard and Suggs (1974) established the genus Exserohilum for *Helminthosporium* species in which the conidial hilium was strongly protuberant, thus providing the third segregate for the graminicolous species presaged by Kenneth, (1958). The new genus was typified by *E. turcicum* (Pass.) Leonard and Suggs.

(ii) Generic Synonymy

The generic name Helminthosporium is deeply entrenched in phytopathological literature and segregation of the graminicolous species into *Drechslera,*

Bipolaris and *Exserohilum* has not been accepted universally.

Subramanian and Jain (1966) argued that accommodation of the graminicolous *Helminthosporium* species in two genera, *Drechslera* and *Bipolaris*, was not warranted. They, therefore, amended the description of *Drechslera* to include all species previously disposed as *Drechslera* or *Bipolaris*. The latter genus and Exserohilum have listed as synonym of *Drechslera* by Ellis (1971, 1976).

(iii) Differentiating criteria for *Drechslera, Bipolaris* and *Exserohilum*

Luttrell, (1963, 1964) suggested additional criteria to those previously used in differentiating *Drechslera* and *Bipolaris,* which might be useful at generic rank. The new criteria were the point of origin and direction of growth of germ tubes from the basal cell of the conidia and the progression of septum development in maturing conidia. In *H. sorokinianum*, the germ tube arises close to the hilum and grows out approximately in the direction of the long axis. This growth was termed semi axial, to distinguish it from axial emergence where the germ tube is percurrent through the hilum, as for example in *Corynespora* (Luttrell, 1963).

In developing conidia of *H. avenaceum*, the primary septum forms near the base; delimiting a cell proximally

that becomes the basal cell of the mature conidium. The first-formed conidial septum in *H. sorokinianum*, in contrast, is near the mid-point of the immature conidium (Luttrell, 1963). Shoemaker, (1962) agreed with the utility of these additional characters for distinguishing *Drechslera* and *Bipolaris*, and added another hilum morphology. In *Drechslera*, it is a flat scar included within contour of the basal cell, while in *Bipolaris*, it is slightly to strongly protuberant (Shoemaker, 1962). The importance of hilum morphology in the generic taxonomy of the group was reinforced by the introduction of *Exserohilum* for species in which the hilum is prominently exerted (Leonard, and Suggs, 1974). Conidial shape, colour, germination behaviors, septum development and hilar form were subsequently considered significant generic characteristics (Luttrell, 1977, 1978).

Additional evidence supporting the recognition of three genera within the graminicolous *Helminthosporia* came from a study, in a range of taxa, of the generic criteria identified previously (Alcorn, 1983). In particular, aspects of conidial germination, septum development and hilum morphology were found useful in assigning individuals to genera, and these characteristics are discussed below and summarized in Table 8.

Table8: Differentiating criteria for *Bipolaris*, *Drechslera* and *Exserohilum*.

Character	*Bipolaris*	*Drechslera*	*Exserohilum*
Conidial shape	Fusoid, obclavate-fusoid rarely truly cylindrical; straight or curved	Commonly cylindrical or obclavate-cylindrical, mostly straight	Fusoid, obclavate-Fusoid or cylindrical; straight or curved
Hilum	Often slightly protruding and truncate	Non-protruding, rounded, well defined intrahilar cavity	Strongly protruding, truncate, often with enveloping "bubble"
Germination	Commonly from one or both polar cells, rarely from intermediate cells.	From intermediate and/or polar cells	Comm ɔnly from one or both polars cells, rarely from intermediate cells
Basal germ tube direction of growth, position of emergence septum ontogeny	Semi axial, close to hilum, rarely lateral	Lateral, midway between hilum and basal septum rarely semi axial from near hilum	Semi axial, close to hilum, rarely lateral
First	Median to submedian	Delimits basal cell	Submedian
Second	Delimits basal cell	Median	Supramedian
Third	Distal	Distal	Median or variable
Conidiogenous nodes	Smooth or verruculose	Smooth	Smooth or verruculose

(iv) Germination

Difference in this criterion, in particular amphigenous or indiscriminate as opposed to polar germination, have been considered important in establishing segregates from *Helminthosporium sensu lato* (Ito, 1930; Nisikado, 1928 and Shoemaker, 1959). The characteristic is quite variable in the type species for *Drechslera, D. tritici-repentis*, being conditioned by such factor as substrate for germination and source of conidia; in some instances germination may be from polar cells only (Alcorn, 1983), characteristic of *Bipolaris. Bipolar* or *monopolar* germination occurs commonly in other species of *Drechslera sensu stricto* (Dennis and Wakefield, 1946; Lam, 1984; Paul, 1971; Scharif, 1961 and 1963). Similarly, amphigenous germination may occur in *Bipolaris* and in *Exserohilum* (Alcorn, 1983; Chaudhuri, 1969; Misra 1973; Reddy and Bilgrami, 1969; and Sivanesan, 1985).

Luttrell, (1963), aspects of basal cell germination are of more important than the number and position of cells that germinate. In *Drechslera,* the germ tube emerges more or less in a median position between the hilum and the septum, and grows at a wide angle (often perpendicularly) to the long axis of the *Conidium's* (Alcorn, 1983and Luttrell, 1963). In *Bipolaris* and *Exserohilum,* the germ tube from the basal cell usually emerges immediately adjacent to the hilum and grows in

the direction of the long axis (semiaxially). The hilum is often displaced laterally due to the close proximity of the growing germ tube. In some species of *Bipolaris* and *Exserohilum*, a small proportion of basal cell germ tubes arises laterally and grows at wide angles, as in *Drechslera* (Alcorn, 1983; Luttell, 1976; & Reddy, 1975).

(v) Other Criteria

Conidial shape, colour and curvature have been suggested as useful criteria in differentiating *Drechslera* and *Bipolaris*. In *Drechslera* the conidia tend to be cylindrical, straight and rather pale; in *Bipolaris* they are often fused, variously curved, and darker (Ito, 1930; Luttrell, 1964; Nisikado, 1928; Shoemaker, 1959 and 1962). In *Bipolaris* and *Exserohilum* species the conidiogenous loci on conidiophores, if well separated, are commonly roughened; in *Drechslera* they are usually smooth (Alcorn, 1983).

Graminicolous species of *Drechslera, Bipolaris, Exserohilum* and *Curvularia* and their teleomorphs, have recently been treated in detail by Sivanesan, (1987). The largest genus in this group, *Bipolaris* has 52 species while *Drechslera* and *Exserohilum* have 23 and 20 species, respectively.

2.3 Epidemiology

Jenns *et al.,* (1982) studied the resistance in maize against leaf blight. The selected 10 inbred lines from the open-pollinated maize variety Jarvis from 51 randomly collected lines to represent a wide range of susceptibility to one isolate each of *Bipolaris maydis* or *colletotrichum graminicola.* Ten isolates of each pathogen were selected for a range of virulence on a maize line with average resistance. Resistance and virulence ratings were based on lengths of lesions that developed on leaves of green house - grown seedlings inoculated with 5 µm -1 droplet of suspensions of known spore concentrations. For each disease the 10 maize lines were inoculated in all possible combinations with the 10-pathogen isolates. The experiment was run 6 times with each pathogen. Analysis of variance for individual trials indicated a significant interaction between maize lines and *B. maydis* isolates in all 6 trials and between maize lines and *C. graminicola* isolates in 4 of 6 trials. For both diseases, the combined analysis over all 6 trials revealed no significant interaction. Apparently the expression of specificity in these host pathogen interactions is variable.

Garraway *et al.,* (1989) studied exposure of maize (*Zea mays L.*) leaves to high temperature stress, *i.e.* 42°C for 6 h before inoculation with *Bipolaris maydis* race T followed by incubation in the dark at 28°C for 24 h, resulted in a significant decrease in peroxidase activity in

both resistant and susceptible isolines compared with the control (leaves not exposed to high temperature stress before inoculation) Also at 48 h of incubation, high temperature stress before inoculation decreased peroxidase activity compared with the control in the resistant but not in the susceptible isoline. Moreover, the level of peroxidase activity in high temperature stress treated and control leaves were significantly lower in the susceptible than in the resistant isoline 48 h after inoculation. Exposure to high temperature stress resulted in a significant increase in electrolyte leakage as well as in sporulation in both isoline. Maize leaf extracts containing peroxidase activity as well as leachates from leaves of both isoline exposed to high temperature stress caused an increase in sporulation *in vitro*. Whereas increased sporulation on maize leaves in response to high temperature stress appeared to be related to increased electrolyte leakage, such a relationship was not found with high temperature stress induced changes in maize peroxidase.

Byrnes *et al,* (1989) studied that southern leaf blight (SLB), caused by race 0 of *B. maydis* [*Cochliobolus heterostrophus)* adversely affected yield of 3 maize hybrids B73 x Mo17, FR27 x Pa 91 and Pioneer 3183. Severity of SLB in mid to late August ranged from 0-5% on FR27 x Pa91 at Tolono, IL to 10-40% on Pioneer 3183 at Urbana. Regressions of yield on area under the curve

and on severity of SLB were significant for 7, 6 and 4 of the 9 locations for Pioneer 3183, B73 x Mo17 and FR27 x Pa91, respectively. Yield of FR27 x Pa 91 and B73 x Mo17 was reduced about 0.7-0.8% for each 1% increase in severity of SLB between 0 and 25%. For Pioneer 3183, yield was reduced about 0.6 - 0.7% for each 1% increase in severity between 0 and 25%. Yield of Pioneer 3183 decreased an additional 23% when severity increased from 25 to 49%. The effects of SLB on yield of maize varied by location, this indicated by differences in slope coefficient from regression models. Unknown factors associated with location may have influenced this relation.

Gowda *et al.*, (1989) studied the incidence of *Exserohilum turcicum* [*Setosphairia turcica*] on the susceptible cultivars CM-202 sown at 2 weeks interval. Disease incidence varies with the sowing data. The incidence was lowest in crops sown in January and February and highest in those of July-September. No disease was observed at 45 days in some crops because of very low RH.

Raziq and Ahmad (1992) studied the effect of different inoculums levels of *Bipolaris maydis* (Nisik.) shoemaker viz. 2 x 10^4, 3 x 10^4 and 4 x 10^4 spores/ml applied at tasselingl, silking and blister stages. They recorded disease severity, yield and yield components of maize variety Azam. The disease severity was the highest

when plants were inoculated with 4 x 10^4 spores/ml at tasseling stage. Grain yield, fresh ear weight, number of Kernels/ear and 200-kernal weight were 28.3, 30.9, 32.5 and 11.8 percent, respectively in this treatment. Plots, where no inoculation was made, showed the lowest disease attack and the highest grain yield and yield component.

Gowda *et al.*, (1993) reported that incidence of *Exserohilum turcicum* [*Setosphaeria turcica*] depended on location, meteorological factors (temperature RH and rainfall), cultivar susceptibility and cultural practices followed by the local farmers.

According to Sharma and Mishra (1993) Turcicum leaf blight of maize caused by *Helminthosporium turcicum* [*Setosphaeria turcica*] is a major threat to the cultivation of maize, especially in Bihar. In a field trial it appeared during the 4th week of November when temperature was 21.1 C and RH 75.2%. Leaf blight was positively correlated with temperature and negatively with RH in crops at all sowing dates, 21 October to 21 December. Disease development was slow in December-January at low temperature and high RH, but inoculums built up during January under conditions of mist and dew, leading to rapid disease development during February and March when environmental factors become more favorable. The progress of the disease could be predicted by the use of a curvilinear bio-meteorological model.

Tsai *et al,* (1993) determined the effects of southern corn leaf blight caused by *Bipolaris maydis* [*Cochliobolus heterostrophus*] on yields. Disease severity and meteorological factors were recorded. The linear regression equations for disease development were obtained by logit [Ln (x/11-x)] transformation of severity with time. However, disease occurrence and development varied with cultivars and locations. In the spring maize, the trends of disease development showed the same tendency but no appropriate equations were obtained for the same cultivars at. Temperature, RH and rainfall, the optimum being 16-32°C and RH 80-100%, irrespective of crop season, cultivars and location, affected disease development. Two regression equations were developed for each cultivars based on the relationship of yield loss to percentage leaf area infected.

Iqbal and Ahmad (1994) assess yield losses and yield components of maize variety. Shaheen after artificial inoculation with *Bipolaris maydis* [*Cochliobolus heterostrophus*], the causal agent of maydis leaf blights. Significant differences (P=0.05) were observed in disease severity and yield between high (2 x 10^4 spores/ml) early (5-6 leaf stage) inoculation and fungicide protected treatments, although losses were also recorded for medium, early, low, early and high delayed inoculations. Disease severity (40.62%), losses in grain yield (29.51%), losses in number of kernels/ear (30.53%) and losses in

200-kernel weight (9.2%) were greatest in the high, early-inoculated treatment.

According to Carson, (1998) selection occurring during the saprophytic or over wintering phase of the life cycle of *Cochliobolus heterostrophus*, the causal agent of southern leaf blight of maize may be a factor in the persistence of apparently less aggressive isolates in the pathogen population. The relative aggressiveness and ability to perennate of 22 isolates of *C. heterostrophus* was measured. Significant differences in aggressiveness and percent premonition (over wintering survival) were observed. There was a weak but often significant negative correlation between the ability of isolates to persist on the soil surface and their aggressiveness. The ability of race 0 isolates to sporulate on senescent corn leaf discs was positively correlated with their aggressiveness. Selection against increased aggressiveness during over wintering does not appear sufficient by itself to counter selection for increased aggressiveness occurring during the pathogen's pathogenic phase.

Pal and Kaiser (2001) studied the Kharif (summer/monsoon) maize (*Zea mays*), which is popular in the eastern part of the country, may be predisposed to maydis leaf blight (*Drechslera maydis* [*Cochliobolus heterostrophus*] Nisikado Race 'O') if proper agronomic practices are not followed. Under the artificial epiphytotic

conditions showed that the disease incidence was favored by planting in July, while early planting in May or June or late planting in August reduced the incidence. Disease incidence gradually increased with the increase in plant density and it was maximum at a population of 70000 ha-1, while it was minimum at 40000 ha-1. Nitrogen alone or in combination with phosphorus and potassium, or with the increase in the dose of nitrogen and the maximum infection occurred at the highest dose of nitrogen at 160 kg ha-1. *In vitro* study, however, showed that nitrogen significantly increased the linear growth of the pathogen, while both phosphorus and potassium individually or in combination with nitrogen reduced it. Nitrogen also significantly increased the percentage of conidial germination, while both phosphorus and potassium individually or in combination with nitrogen reduced it.

2.4 Resistance

Levy (1989) reported that pathogenic fitness and environmental conditions are very important in determining severity as the epidemics depend on the ability of *E. turcicum* to infect, grow and sporulate on maize plants. In the United States the disease has been effectively controlled by the use of the dominant *Ht* gene [Hooker (1961), Smith and Kinsey, (1980), Turner and Johnson, (1980)]. A new choloratic halo gene for resistance of limited commercial value but which may be

useful in combination with Ht genes has been reported (Carson, 1995a)., Pratt *et al.* (1993) reported a polygenic based resistance in Ohsio expressed as rate reducing resistance or low number of lesions using a 0-5 severity rating scale. Combining *Ht 1* and *Ht 3* genes did not result in significantly less disease from those homozygous for each *Ht 1* or *Ht 3* (Leath and Pedersen, 1986). However, Dunn and Namm (1970) reported gene dosage effects for the *Ht* gene, and Hooker and Perkins (1980) reported gene dosage effects for the *Ht 2* gene. Smith and Kinsey (1980) suggested that a combination of *Ht 1* and *Ht 2* or *Ht 3* would confer resistance against race 1, 2 and 3. Pataky (1994) showed that high levels of partial resistance with or without *Ht* genes presented a spectacular approach reducing damage from northern leaf blight on sweet corn, which also eliminates the severe yield depressing chlorosis associated with *Ht* gene resistance in very susceptible backgrounds. Carson (1995b) indicated that the latent period is related to partial resistance, which suggested that selection for increased latent period length would be more beneficial than selecting for reduced disease severity. Selection for increased latent period length can be done in environments without severe disease epidemics and also breeding material could be assessed as seedlings for latent period length in the green house during the off season. Levy (1991) showed that isolates from different areas were different in parasitic fitness as was indicated

by infection efficiency, sporulation and lesion size while isolation from the same location showed less variation. Inoculums in previous crop have been found to be critical in epidemic build up for subsequent cropping especially in non-tillage systems as reported by Pedersen and Oldham (1992) using race 2. Pataky (1992) found that yield losses were significant when disease severity was high on the upper leaf canopy which is in agreement with the studies of Levy and Leonard (1990), Raymundo (1978) and Solomonovich *et al.*, (1992) who found that plants defoliated of the lower third of all the leaves showed no yield losses. Leath and Pedersen (1986) found that a cross between resistant B 37 *Ht* 3 and susceptible B 37 had a severe chrolosis associated with resistant lesions resulting in a high area under the disease progress curve (AUDPC) value for resistant inbred with low sporulation and secondary spread. One biotype is a virulent to lines carrying genes *Ht 1, Ht 2, Ht 3* and *Ht N.* The other biotype is a virulent to lines with genes *Ht r. Ht 3* and *Ht N* but is virulent to maize carrying genes *Ht 1 A* or *B* (Shurtleff, 1973).

Classification of isolates of *E. turcicum* into races is based on the resistant genes marked by an isolate in the widely used nomenclature as suggested by Leonard *et al.* (1989). They proposed that evaluations be carried out in temperatures near 20°C and light intensities of 20 to 50 lux because reactions associated with *Ht 1, Ht 2* and *Ht 3*

are thermal and photo sensitive. This was demonstrated by Leath and Pederson (1986) when they showed that resistance in lines with *Ht 2* and *Ht 3* was expressed clearly in controlled environment chambers at 22°C and 18°C night temperatures. Race 0 has the resistance formula; *Ht 1, Ht 2, Ht 3, Ht N/*, race 1, *Ht 2, Ht 3, Ht N/Ht 1*; race 2 *Ht 1, Ht 3, Ht N/Ht 2*, race 3, *Ht 1/Ht 2, Ht 3, Ht N*, race 12, *Ht 3, Ht N/Ht 1, Ht 2*, race 23 as *Ht 2, Ht 3/Ht 1, Ht N* race 23 N as *Ht 2, Ht 3, Ht N/Ht 1.* This classification left room for the accommodation of new races that could be encountered in future studies. The *Ht 1, Ht 2* and *Ht 3* resistance occurs as chlorotic lesions with minimum sporulation, while the *Ht N* induced resistance is expressed as a delay in disease development until after pollination (Leonard *et al.,* 1989). Bergquist and Masias (1974) reported the first race of *E. turcicum.* Lipps and Hite (1982) reported the presence of race 1 in Ohio and were virulent on *Ht* and *Ht 1* but a virulent on *Ht 2.* Turner and Johnson (1980) reported a similar race in Indiana. Smith and Kinsey (1980) reported a new race designated race 3 with a virulent formula *Ht 1/Ht 2, Ht 3.* Thakur *et al.,* (1989) reported the presence of yet another race named race 4. Jordan *et al.,* (1983) reported the occurrence of races 1 and 2 from seven states in the Central and Eastern USA where race 1 was virulent on B 37 only and race 2 virulent on B 37 *Ht*; no isolate was found virulent on B 37 *Ht 2* or Oh 43 *Ht 3.* A report by Welz *et al.,* (1993) indicated the

presence of race 0 and race 1 in China; race 23 N, 23 and 2 N in Mexico; race 23, 23 N and race 0 in Zambia; and race 0, N, 23N and race 2 in Uganda.

According to Gevers (1975) the *Ht N* major gene of resistance derived from the Mexican maize variety Pepitila is reasonably stable, but in some parts of the world the effects may fail to be expressed. Genetic segregation may not behave like expected of dominant genes ratios, but does, however remain in the tolerable limits of deviation of stability and segregation. He suggested the occurrence of biotypes in India which were able to overcome the *Ht N* gene of resistance. The *Ht N* gene in some backgrounds was sensitive at high temperatures symptom expression was reduced on B37 Ht3. Plants was evaluated at both 26°C day/22°C night and 22°C day/18°C night temperatures. There was observable weakened virulence at high temperatures. Another isolate from Hawaii was found to cause disease on B 37 HtN, Oh 45 HtN, B 14 A *Ht N* and B68 *Ht N* and was designated race 2N with a virulent formula *Ht 1, Ht 3/Ht 2* (Windes and Pedersen 1990). Pataky *et al.*,(1986) reported that hybrids with or without *Ht 2* did not show significant differences in disease severity induced by races 1 and 2, which may have been due to shading of lower leaves because resistance may be reduced at low light intensities. Recent studies in Uganda by Adipala *et al.*,(1993a) showed that *E. turcicum* occurred in all maize

growing areas and was more severe in wet areas. However, all isolates tested were virulent on A 619 *Ht N*, hence were classified as race 0. These observations are at variance with the results in Uganda by Welz *et al.* (1993). Average disease severity ranged between 0.5 to 25% in Uganda. After evaluations of Ugandan maize germplasm, Adipala *et al.*, (1993b) also reported that all had necrotic susceptible reactions when inoculated with race 0, 1, 23 and 23 N and did not express symptoms typical of the *Ht* gene. Seedling inoculation was useful to identify chlorotic resistance, while adult plants were useful in assessing rate reducing resistance.

2.5 Genetics of Resistance

Hooker (1961) reported the unique chlorotic lesion type of resistance on maize lines characterized by chlorotic lesions, and late developing lesions with small necrotic center surrounded by a light green margin. These lesions produced fewer spores compared to the rapidly developing necrotic susceptible lesions. This type of resistance was found to be control by a single dominant gene Ht. Homozygous dominant plants rarely have lesions. Ullstrup (1963) reported similar results on line P.I. 217407 where small lesions were surrounded by chlorotic halos with very limited sporulation in resistant genotypes. Further work by Hooker (1963) also concluded that a single dominant gene in the dent corn

inbred line GE 440 conditioned the resistant chlorotic lesion type.

Hilu and Hooker (1963 and 1965) showed that symptoms were similar for susceptible and resistant lines, from 2-7 days, which appeared as minute white to light green flecks after inoculation of inbred and hybrid seedlings of GE 440 with *E. turcicum*. On susceptible lines these flecks developed into lesions that wilted before developing necrosis. No wilting was seen on resistant cultivars. Disease development may take about 15 days. Sporulation was delayed 50-80 hrs and the population of spores per unit area may be reduced 60 times in the resistant lesions as compared to susceptible lesions. This is a situation normally seen in monogenic chlorotic lesion resistance but not in multigenic resistance. Ceballos *et al.*, (1991) reported that development of new races shorten the durability of the chlorotic resistant reactions which are controlled by single monogenic resistance genes. Polygenic resistance is normally expressed by reduced number of lesions and decrease in lesion size and amount of sporulation (Ullstrup 1970). Singulas *et al.*, (1988) reported that the average level of resistance, mean lesion area, the rate of increase in lesion size and the shape of the lesion are strongly influenced by host gene make up as determined by contributions of each parent.

Dey *et al.*, (1989) studied that diallel analysis of 8 diverse maize inbred lines for maydis leaf blight resistance (*D. maydis* [*Cochliobolus heterostrophus*] revealed that the general combining ability (GCA) component is more important than the specific combining ability (SCA) component in the inheritance of resistance. The resistant lines generally had lower GCA effects and the crosses between moderately resistant and susceptible lines had negative SCA values. Inbred line Vijay 444 was the best general combiner for resistance to *C. heterostrophus*.

Inoue *et al.*, (1989) studied resistance to *B. maydis* [*Cochliobolus heterostrophus*], banded leaf and sheath spot (*Cercospora sorghi*] Smut [*Ustilago zeae*] and lodging, days to mid silking, spring vigour, tiller number, stalk length and ear insertion height in 376 local Japanese varieties of Caribbean flint type maize. Resistance to *C. heterostrophus* was highly variable, greatest resistance being found in materials from Japan.

Horner (1990) studied that a maize population produced by selecting for resistance to race 0 of *Bipolaris maydis* [*Cochliobolus heterostrophus*] in a wide range of accessions was divided into 2 highly resistant subpopulations, P58A and P58B, on the basis of pedigree records, such that the relationship between the populations was minimized.

Kaiser and Prodhan (1990) ninety-three elite hybrids and composites were tested for resistance to *Exserohilum turcicum* [*Setosphaeria turcica*], *Drechslera maydis* [*Cochliobolus heterostrophus*] and *Physoderma maydis*, using standard whorl inoculation, India. The genotypes varied greatly in their reactions to the diseases, with the average grade ranging from resistant to highly susceptible. Among the genotypes tested, multiple resistances to the 3 diseases were recorded in Philippine DMR Composite. Philippine DMRI, Pratap Composite, EVA (MDR2) 76, MCU204 and MCU216 and an acceptable level of resistance was provided by Philippine DMR2, Philippine DMR5, EVA (MDRI) 76 and Mg.

Liu *et al.*, (1991) through field and laboratory experiments showed that the sub group C 1 (CMS-C) of group C maize was especially susceptible to *Bipolaris maydis* [*Cochliobolus heterostrophus*] race C, but sub groups C 11 (CMS-RB) and C 111 (CMS-ES) were not seriously infected.

Mahajan *et al.*, (1991) evaluated twelve diverse maize cultivars for resistance to *E. turcicum* [*Setosphaeria turcica*]. Meghalaya Local yellow, a susceptible control, was used as the infector row for uniform spread of the disease. Natural disease incidence was recorded at the dry-silk stage. Deccan 103, Suwan 1, Ganga 11 and Hemant were classified as resistant,

and NLD DFP 70 and VC 80 x (Eto tuxp br2) as moderately resistant. The severity of the disease did not differ significantly, but incidence was least for the crop sown in April.

Ali and Ahmad (1992), evaluate ten maize genotypes i.e. Azam, Bahar, Ehsan, Peshawar local, PS 7930, PSVE-II, PSEV-4085, Sarhad white, Sarhad yellow and Shaheen for their relative reaction to maize leaf blight and grain yield under field conditions. None of the test cultivars exhibited complete resistance, to the disease. However, genotypes PSEV 4085 and Sarhad yellow showed low reaction to the disease and the maximum yield of 5836 and 5689 kg/ha, respectively. Variety Bahar was the most susceptible and gave the lowest yield of 3784 kg/ha.

Aziz, *et al.*, (1992) tested eleven Pirsabak hybrids of maize against two approved synthetic/open-pollinated (OP's) maize varieties under irrigated condition. Data showed that PSH-10 was resistant to stalk rot and leaf blight (*Helminthosporium maydis* and *turcicum*) diseases. Sarhad yellow (Check-1) although mediocre in yield, was susceptible to stalk rot and foliar disease.

Gowda *et al.*, (1992) studied combining ability analysis of 6 maize parental lines for *Exserohilum turcicum* [*Setosphaeria turcica*] infection indicated that CM-118, CM-104 and Ade Cuba were good general

combiners for resistance and imparted high specific combining ability into cross combinations. Two single crosses exhibited maximum heterosis for disease resistance.

Sain Dass *et al.*, (1992) were grown and evaluated six maize parents (2 highly susceptible, 2 moderately resistant and 2 resistant) and they are 10 F 15, for resistance to *Drechslera maydis* [*Cochliobolus heterostrophus*] under conditions of natural infection. Disease incidence was recorded at 60 days and at maturity and pooled data analyzed. Low susceptibility was dominant over high, with the best resistance obtained in hybrids involving resistant and moderately resistant parents in different combinations. The absence of complete resistance in either parents or hybrids indicated that various interactions of polygene are involved in the inheritance of the disease.

Dey *et al.*, (1993) were evaluated eighteen composites and hybrids of maize in the field in Panjab, India, for resistance to the pyralid *Chilo partellus* and *Drechslera maydis* [*Cochliobolus heterostrophus*] and *Sclerophthora rayssiae* var. *zeae*. Multiple resistances were scored on the basis of genotypic score and standard deviation for a genotype over the 3 stress factors. Four genotypes were found resistant to *C. heterostrophus*. J684 HSC3, J115 (Parbhat) and J54ZFS4 showed resistance to both diseases. J115 (Parbhat) also had a

comparatively higher level of multiple resistances when diseases and pyramid were considered together.

According to Chang and Peterson (1995) *Bipolaris* (*Helminthosporium*) maydis is the causal fungus of southern leaf blight of maize. Resistance to this disease has been determined to be control by a single recessive gene, designated *rhm*. The dominant allele, *Rhm*, confers susceptibility to the fungus. In one project to tag the *rhm* gene with transposable elements, *Rhm/Rhm EIEI* (Eielement) lines were crossed to an *rhm* tester. Screening for mutation to *rhm* was then conducted on the F1, *Rhm/rhm* E1, and seedling. Element insertions into *Rhm* are expected to be random events and will mutate *Rhm* phenotypically to *rhm* (susceptible to resistant). In these tests mutation rates of *Rhm* to *rhm* were usually in the order of 10 (-5). However, hybrid populations between to different *Rhm* E1 lines (Cy line and T line) yielded approximately 5% mutants. To account for the unexpectedly high mutation rate, a hypothesis is proposed that there is two linked receive genes controlling resistance. The two lines combined differed in genotypic content and the unexpected 5% mutants arose from cross over between the two dominant alleles at the two linked loci in repulsion phase (*Rhm 1 rhm2/rhm1 Rhm2*). It is also postulated that one of the two genes is a copy of the other created by duplication. The dominant status at either locus makes a

functional product and thus abolishes resistance. The two-gene model is currently being tested. The significance and implications of this finding are discussed.

Chang and Peterson (1995) a recombination test showed that the maize lines Cy and T carried the *B. maydis* [*Cochliobolus heterostrophus*] Chlorotic lesion resistance genes *Rhm1* and *Rhm2*, respectively. These designations are reversed from previous reports.

Hipskind *et al.*, (1996) a final stage of resistance expression involving the accumulation of a pigment in uninfected, healthy epidermal cells surrounding restricted lesions on leaves of maize resistant to *B. maydis* [*Cochliobolus heterostrophus*] is described. The pigment was characterized by plasma desorption mass spectrometry and had a structure consistent with that of Cyanidin 3-dimalonyl glucoside. The function of the zwitterionic anthocyanin involves the protection of uninfected healty plant tissue from the toxic, oxidative metabolites that accumulate during the expression of resistance. Microscopic examination of the leaf tissue revealed that the pathogen stopped growing in the resistant cultivar 18 hrs post infection, considerably prior to pigment accumulation. The pigment, therefore, accumulated in cells that were affected but not infected by the pathogen.

According to Lambert and White (1997) future maize (*Zea may L.*) productivity increases require breeding materials with high yield potential and multiple disease resistance. As part of an integrated program to develop breeding populations with high grain yield potential and multiple disease resistance, two maize synthetics were reciprocally recurrently selected for yield and mass selected for multiple disease resistance. The objective of this study was to determine selection response of two maize synthetics to six cycles of tandem selection for multiple leaf disease (MLD) and multiple stalk rots (MSR). Plants were inoculated each cycle and evaluated for MLD including their causal agents; northern corn leaf blight (NCLB) [*Exserohilum turcicum* (*Pass.*) Leonard and Suggs, Race 0 and 1], southern corn leaf blight (SCLB) [*Bipolaris maydis* (Nisik) shoem.], northern corn leaf spot (NCLS) [*Bipolaris zeicola* (sotout) Shoem. Race 1, 2 and 3 anthracnose leaf blight [*Colletotrichum gram*inicola (CES) G.W. Wils.] and eyespot (*Kabatiella zeae* Narita and Hirzatsuka). Following anthesis, plants were inoculated and evaluated for resistance to MSR including their causal agents; *Diplodia* stalk rot (DSR) [*Stenocarpella maydis* (BERK) Sutton = Syn. *Diploidia maydis* (BERK)], anthracnose stalk rot (SASR) (*Colletotrichum graminicola*), G*ibberella* stalk rot (GSR) [*Gibberella zeae* (Shw.) Petch] and Fusarium stalk rot [*Fussarium moniloforme*, shield]. In 1993 and 1994, selection cycles 0, 2, 4 and 6 of synthetics RSSSC,

RB510 and their cycle crosses were evaluated. Selection response to MLP, NCLB, SCLB, NCLS, gray leaf spot GLS; (*Cercospora zeae maydis* Jehon and Daniels), MSR, DSR, GSR and ASR were measured in separate experiments. Decreases in leaf blight severity from C0 to C6 in RSSSC was 29% for MLD, 23% for NCLB, 33% for SCLB, 28% for NCLS and 21% for GLS. Decreases for RB 510 were 34% for MLD, 33% for NCLB, 37% for SCLB, 49% for NCLS and 16% for GLS. Cycle crosses were usually intermediate in values for leaf blight red.

Liu *et al.*, (1997) studied seedlings of 10 cultivated inbred lines of maize, which were, inoculated with new isolates of *Bipolaris maydis* [*Cochliobolus heterostrophus*] in the green house to monitor the progress of race 0 as the predominant isolate. Resistance to *B. maydis* has been gradually lost in new cultivars due to the increase of race 0 among pathogen populations.

According to Collins *et al.*, (1998) many of the plant disease resistance genes that have been isolated encode proteins with a putative nucleotide-binding site and Leucine rich repeats (NBS-LRR resistance genes). Oligonucleotide primers based on conserved motifs in and around the NBS of known NBS-LRR resistance proteins were used to amplify sequences from maize genomic DNA by polymerase chain reaction (PCR). Eleven classes of non-cross hybridizing sequences were obtained that had predicted products with high levels of amino

acid identity to NBS-LRR resistance proteins. These maize resistance gene analogs (RGAs) and one RGA clone obtained previously from wheat were used as probes to map 20 restriction fragment length polymorphism (RPLP) loci in maize. Some RFLPs were shown to map to genomic regions containing virus and fungus resistance genes. Perfect co-segregation was observed between RGA loci and the rust resistance loci *rp1* and *rp3*. The RGA probe associated with *rp1* also detected deletion events in several rp1 mutants. These data strongly suggest that some of the RGA clones may hybridize to resistance genes.

According to Morris *et al.*, (1998) Systemic acquired resistance (SAR) is a widely distributed plant defense system that confers broad-spectrum disease resistance and is accompanied by coordinate expression of the so called *SAr* genes. This type of report that chemical inducers of resistance are active in maize. Chemical induction increases resistance to downy mildew and activates expression of the maize *PR-1* and *PR-5* genes. These genes are also coordinately activated by pathogen infection and function as indicators of the defense reaction. Specifically after pathogen infection, the *PR-1* and *PR-5* genes are induced more rapidly and more strongly in an incompatible than in a compatible interaction. In addition, monocot lesion mimic plants also express these defense related genes and that they

have increased levels of salicylic acid after lesions develop, similar to pathogen-infected maize plants. The existence of chemically inducible disease resistance and *PR-1* and *PR-5* gene expression in maize indicates that maize is similar to dicots in many aspects of induced resistance. This reinforces the notion of an ancient plant inducible defense pathway against pathogen attack that is shared between monocots and dicots.

Namjeet *et al.*, (1999) were developed non-conventional inter-synthetic hybrids using factorial mating design and evaluated in randomized complete block design. The line x tester analysis indicated prevalence of both additive and non-additive gene effects for resistance to *Bipolaris maydis* [*Cochliobolus heterostrophus*] with predominance of non-additive gene effects. Narrow based synthetics NBS JE63, NBS A68 and NBS TUX. EP was good general combiners for lower incidence of the disease. The inter-synthetic crosses possessed significant negative SCA effects for disease resistance.

According to Welz and Geiger (2000) turcicum or northern corn leaf blight (NCLB) incited by the *Setosphaeria turcica*, anamorph *Exserohilum turcicum*, is a ubiquitous foliar disease of maize. Diverse sources of qualitative and quantitative resistance are available but qualitative resistances (*H1* genes) are often unstable. In the tropics especially, new virulent races either overcome

them or they suffer from climatically sensitive expression. Quantitative resistance is expressed independently of the physical environment and has never succumbed to *S. turcica* patho types in the field. This review emphasizes the identification and mapping of genes related to quantitative NCLB resistance. We deal with the consistency of the genomic positions of quantitative trait loci (QTL) controlling resistance across different maize populations and with the clustering of genes for resistance to *S. turcica* and other fungal pathogens or insect pests in the maize genome. Implications from these findings for further genomic research and resistance breeding are drowning. Incubation period (IP) and area under the disease progress curve (AUDPC) based on multiple disease ratings, are important component traits of quantitative NCLB resistance. They are generally tightly correlated (r (P) approximately equal to 0.8) and highly heritable (*H2* approximately equal to 0.75).

According to Kraja *et al.,* (2000) a possible use of non-elite germplasm is as a source of alleles for disease resistance.

Sain-Dass *et al.*, 2000 were studied the gene effect for maydis leaf blight (*Drechslera maydis* [*Cochliobolus heterostrophus*]) disease resistance and grain yield in exotic and indigenous white maize grain (*Zea mays*) from line x tester analysis. Combining ability analysis revealed

that non-additive genetic variances were more important in the expression of disease resistance and high grain yield. There was no similarity in ranking between per se performance and its corresponding SCA effects of the crosses. In general, it was found that resistance level of the crosses to maydis leaf blight disease was increased where both the parents were having disease reaction resistant/least susceptible followed by resistant x susceptible irrespective of incidence of male parents.

Sain-Dass *et al.*, (2002) conducted combining ability analysis for disease resistance and yield in Karnal, India, using ten maize lines and five testers along with 50 resultant hybrids and 5 controls. The experiment was conducted in 3 environments: natural disease (caused by *Drechslera maydis*) conditions and early planting (30 June) before the onset of monsoon, artificial disease conditions and early planting before the onset of monsoon and artificial disease conditions and late planting after the onset of monsoon. Combining ability analysis revealed the importance of general combining ability (gca) and specific combining ability (sca) variances, with sca being more pronounced than gca variances for yield and disease traits. Gca revealed that KI 628, KI 682, KI 701, KI 856 and CML 348 were most desirable as they contributed maximum favorable genes for disease resistance and high yield. CML 340 x KI 82, CML 228 x KI 708, KI 112 x KI 708 and KI 682 x KI 202

were superior under natural and artificial disease conditions due to their high specific combining ability effects.

Robbertse *et al.*, (2003) detected the *Cochliobolus hetrostrophus* monofunctional catalase - encoding genes and reveal its role in virulence.

Degani *et al.*, (2004) and Ganem *et al.*, (2004) demonstrated the role of G. protein beta subunit of *C. heterostrophus* in virulence, asexual and sexual reproductive ability and morphogenisis.

Nonrebosomal peptides, made by nonribosomal peptide systhetases, have diverse biological activities, including roles as fungal virulence effectors. Inspection of genome of *C. heterostrophes* the fungal pathogen of maize and a member of a genus noted for secondary metabolite production, revealed eight multimodular non ribosomal peptide synthetase/ polypeptide synthetase hybrid enzyme presumed to be involved in anthesis of a peptide / polypeptide molecules. Deletion of each NPS gene and phenotype analysis showed that the product of only one of these genes, NPS 6, is required for normal virulence on Maize (Lee et *al.*, 2005).

A total of 11 quantitative trait loci (QTLs) were found to condition resistance to SCLB/ SLB depending upon which disease ratings were used in the analysis. When the AUDPC data were combined and analyzed over

environment, seven QTLs, on Chromosomes 1, 2, 3, 4, 7 and 10 were found to come from resistant parent Mo 17. Additional QTLs for resistance on Chromosome 1 came from the susceptible parent B 73. The eight identified QTLs accounted for 46% of the phenotypic variation for resistance (Carson *et al.*, 2004).

MATERIALS & METHODS

Chapter- 3

MATERIALS AND METHODS

3.1 General Precautions

The glassware's used in the present studies were of Borosilicate glass. These were thoroughly washed with chromic acid and tap water and rinsed with distilled water and kept in hot air oven at 160°C for two hours and were stored in oven.

The seeds were surface sterilized with 4 per cent solution of Sodium hypochlorite (NaClO) solution for 2 minutes and washed 3 to 4 times with sterilized water.

3.2 Survey for prevalence and severity of disease in different locality

Maydis leaf blight of maize (*Zea mays*) is a common maize disease. This disease is widely spread in north and

south India. The disease is also found in and around Jhansi district.

The survey for this disease was made in Kharif of 2003 and 2004. During surveys, twenty plants were randomly selected and incidence & severity was noted.

Infected maize samples were collected from various part of Jhansi. A minimum of ten samples was collected from each location. The samples were stored in polythene bags and brought to the laboratory for further investigations.

3.3 Isolation of Pathogens

Piece of infected maize leaves having disease symptoms sterilized with 0.1 per cent mercuric chloride and transferred to sterilize petri plates containing 10 ml PDA. The isolation was made inside and laminar flow, which was pre-exposed to UV radiation. The petri plates were kept in BOD incubator at 22 ± 1°C for a suitable period for growth of pathogen. The isolated pathogen revived at 7-10 days interval for further study.

The Potato Dextrose Agar (PDA) medium was prepared by taking 200 gm pealed and sliced potatoes, extracting it in water. The extract was added with 20 gm each of Dextrose and fine powdered Agar and making the volume to 1000 ml. The media was sterilized in an autoclave at 15 lbs pressure for 25 minutes.

3.4 Mass multiplication & Inoculation Techniques

Method-I: Original cultures of the pathogen were isolated from leaf lesions by placing in moist chamber. After five days newly formed spores on the surface of the lesions were picked up with a fine flattened needle under a dissecting microscope, placed in a droplet of sterile water and streaked across the surface of hardened, acidified water agar in Petri plates. After allowing a few hours for the spores to germinate, they were cut out of the agar and transferred to hard, acidified potato dextrose agar. After two weeks of incubation at 20-25°C, these cultures were transferred to fresh plates of acidified potato dextrose agar for multiplication. When the fungus growth has covered the surface of the agar the cultures are ready for use. About 20 petri plates of culture are macerated in water in a warring blender for 15-30 seconds, strained through a layer of cheese or muslin cloth and made up to four five liters of suspension. This stock suspension was taken to the field and diluted in a compressed air sprayer at the rate of one liter in 12 liters of water.

Inoculums should be directed into the whorls of the plants, where it will be retained long enough to permit spore germination. Inoculations repeated thrice when plants are 30-45 cm high. This method has been uniformly successful in establishing heavy infection so that resistant plants could be selected before silking.

This method was used for studying disease resistance in maize genotypes.

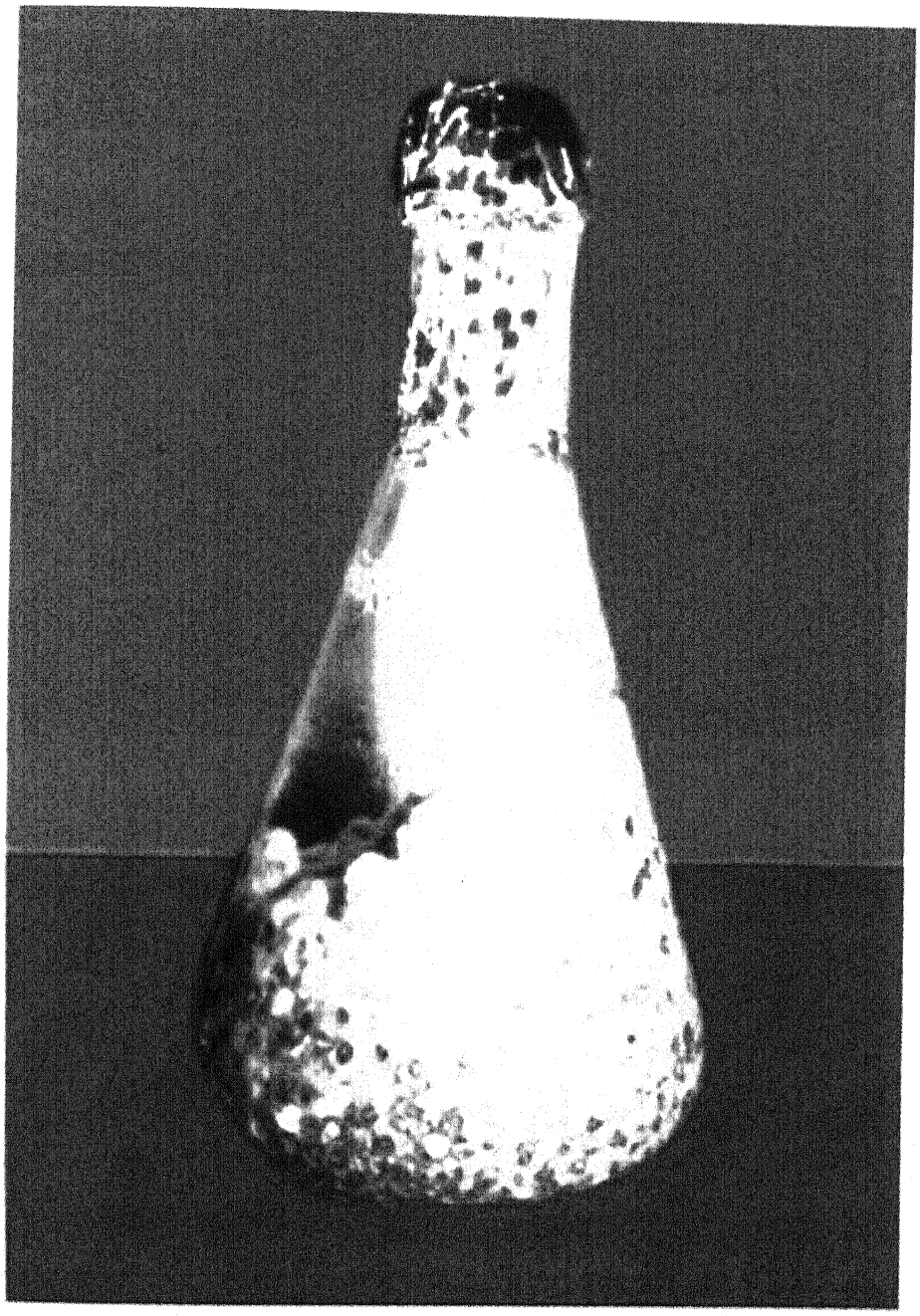
Method-II: This procedure followed for identifying reoccurrence of disease inoculums are most easily carried out by gathering of heavily infected leaves of previous year. This should be done before leaves become fully mature. Leaves were stored in large gunny bags in some dry room protected from moisture. Just before inoculation, the dry leaves are ground into a meal of about the coarseness of wheat bran.

Inoculation was done by placing a pinch of leaf meal (a heaped thimbleful), into the whorl of each plant, when crop was 30-45 cm high. A second inoculation was made five to ten days later.

Method-III: Grain (sorghum seed), cultures of the blight pathogen were started three weeks before planting the test material. When the pathogen had grown over the grain Fig (4), grind it in a food chopper and store at 6-9°C. At the time of inoculation, this inoculum was diluted to the desired level by adding ground sorghum seeds.

Inoculation was done by placing a small quantity of pathogen into the whorl of each plant. Inoculation was normally made on a cloudy day or towards evening to avoid mortality by direct exposure to sunlight.

Fig. 4: Mass multiplication of *D. maydis* on sorghum seed.



3.5 Pathogenicity Test

Pathogenicity of the isolated fungus was tested following Koch's postulates on maize genotype raised in earthen pots in net house conditions. The air-dried sorghum grains with fungal growth were used for inoculations on 30 days old plants by inserting 3 to 4 grains gently leaf sheath of each plant. High humidity was maintained during disease development by frequent irrigations. Symptoms appeared with 3-4 days after inoculation of the maize plant. These could be observed on leaves. Symptoms initiated as elliptical, water soaked, straw-coloured lesions on leave.

3.6 Biology of the Pathogens

After incubation period of 7 days, a temporary mount was prepared in Lecto-phenol and cotton blue from various isolates collected from different location and examined under microscope for their shape and size of conidia and conidiophores.

Conidial germination or growth character was observed on grease free, glass slide thinly coated with conidia in a drop of sterilized distilled water. The slides were kept in moist chambers made of petri plates prepared by putting 2-folds of what man's filter paper in both sides of petri plate covers. These petri plates were incubated at 27°C. After three days conidial growth were studied.

The size of the conidia was measured using ocular and stage micrometer. The number of division of stage micrometer coinciding with divisions of the ocular micrometer were noted the ocular index (μ) was calculated by the formula as follows

$$\text{Ocular index } (\mu) = \frac{\text{Number of division of stage micrometer coinciding with Ocular division}}{\text{Number of division of ocular micrometer coinciding with Stage division}} \times 10$$

Where, ten in the above formula is the constant multiple, as one division of stage micrometer = 10 μ.

3.7 Germination behaviour

The conidial germination was studied by hanging drop method. The moisture plate was incubated in BOD Incubator at different temperatures. After incubation of 24 hours germination behaviour was observed. Besides, times taken for 1 cm growth of germ tube at different temperature were also recorded. The development of hyphal length, axial hyphal and number of hyphal cells at different hours were also made by same procedure.

3.8 Growth of *D. maydis* on selected media

Pure culture of *D. maydis* maintained on PDA was used for studying the growth in different media. The selected media and their composition are as:

Corn meal Agar

Corn meal	20 gm
Agar	20 gm
Glucose	20 gm
Peptone	20 gm
Water	1000 ml

Oat meal Agar

Oatmeal	30 gm
Agar	20 gm
Water	1000 ml

Malt extract Agar

Malt extracts	20-50 gm
Agar	20 gm
Water	1000 ml

Czapek's Dox Agar

Sodium nitrate	2.0 gm
Potassium monohydrogen phosphate	1.0 gm
Magnessium sulphate	0.5 gm
Glucose	20 gm
Agar	15 gm
Water	1000 ml

Potato Dextrose Agar

Peeled Potato	200 gm
Dextrose	20 gm
Agar	20 gm
Water	1000 ml

Richard's media

Potassium nitrate	10 gm
Potassium dihydrogen phosphate	5 gm
Magnessium sulphate	2.5 gm
Ferric chloride	0.02 gm
Succrose	50 gm
Agar	15 gm
Water	1000 ml

The media were prepared as per recommended procedures. The inoculations and incubations were also made as per the procedure described earlier. There were three replications for each treatment. Observations were recorded on 3rd, 7th and 10th day on the colour of mycelium, colour of the media at the growth point, mycelium emergence and growth of the fungus.

3.9 Study of the optimal temperature for the pathogen:

The effect of different media and temperature of the pathogen were studied under laboratory conditions. The colony of *D. maydis* were grown on PDA in Petri plates, when colonies filled the Petri plates, 4 mm diameter

mycelial piece/disc, cut from colony margins, were transferred to the center of 90 mm Petri plates, each containing 20 ml media. For mycelial growth, six different commonly used synthetic and sterilized solidified media *viz.*, Corn meal Agar, Oat meal Agar, Czapek's Dox Agar medium, Potato Dextrose Agar medium, Richard's medium and Malt Extract Agar were examined under incubated conditions at 20°C, 25°C, 30°C, 35°C and 40°C temperature. The diameter of the resulting colonies was measured after 7 days of incubation.

The growth of *D. maydis* (colony diameter) was measured with the help of Hi Antibiotic zone scale.

3.10 Mode of Infection and perpetuation of the pathogen

(i) Role of Seeds: When the disease was at very high intensity then all plant parts were infected. So knob or seed of the plant were also infected. Seeds were collected from diseased plants and stored under room conditions. During next season, these collected seeds were sown in 10" diameter earthen pots with sterilized soil in net house. After 4-5 days, germination started and propagules emerged out from soil. The germinating propgules were watched for the presence or absence of disease.

(ii) Role of infected soil: For studying role of infected soil in disease epidemics, ten surface sterilized healthy seeds were sown in naturally infected field soil, filled in 10" diameter earthen pots. After germination, plants were constantly watched for disease appearance.

(iii) Role of diseased plant debris: It is evident that the diseased plant debris work as inoculums. These plant parts left or fallen in fields infect healthy plants. The role of plant debris in disease spread was done in pot under net house condition. Diseased plant debris /leaves wrapped in tissue paper and were stored at room temperature in the laboratory. During next season the plant debris were tested through TTC test and also grinded and sprayed on the whorl of healthy plants and observed for the disease occurrence.

(iv) Secondary spread through air: The healthy plants of maize were raised in pots by sowing sterilized seeds in sterilized soil. Diseased plants of the maize were also raised in pots and placed near the healthy ones and observe for disease occurrence.

(V) TTC Test: The dried infected leaves were collected in paper bags and stored at room temperature. During the next season, a portion of collected materials was soaked in distilled water and later incubated at 30°C for 48 hours. The soaked materials were treated with T.T.C. solution (1% solution of 2, 3, 5-triphenyl tetrazolium

chloride) prepared in buffer pH 6.5 - 7.0 [Buffer had been made by adding 2 part of solution A (9.078 gm KH_2PO_4 /l distilled water) and 3 part of solution B (11.876 gm Na_2HPO_4. $2H_2O$ /l distilled water)] and again incubated at 30°C for 48 hours in darkness (Pathak *et al*, 1978).

After incubation, temporary mount was prepared in distilled water and observed in light microscope. Living pink coloured mycelium or conidia will be seen.

3.11 Disease or Field Reaction

The disease was observed at mid whorl collar of 8th leaf visible, first and second leaf dead (Table 9, Fig. 5). Rating was done on scale consisting of five broad categories designated by numerals 1 to 5 as proposed by Payak & Sharma (1983). Wherever possible observation, on lesion type were also made such as large sporulating type or small chlorotic and non-sporulating type. The rating scale is as following:

1.0 Very slight to slight infection, one or two to few scattered lesions on lower leaves.

2.0 Light infection, moderate number of lesions on lower leaves, few on middle leaves.

3.0 Moderate infection, abundant lesion on lower leaves, few on middle leaves.

Table 9: Growth stages of maize crop.

Stage	Description
0	Pre-emergence
1	Emergence, coleoptiles above soil
2	Single leaf open
3	Two to three leaves fully open
4	Early whorl, collar of fourth leaf visible, nodal roots developing, leaf growth
5	Mid-whorl, collar of 8th leaf visible; 1st or 2nd leaf may be dead, leaf growth
6	Late whorl, collar of 12th leaf visible, 1st to 4th leaves may be dead, leaf growth
7	Tassel tips of the tassel visible
8	Silk, silk visible, pollen shedding
9	Crop maturity
9.1	Cob full size, kernels in blister stage
9.2	Kernels in "Soft dough" stage
9.3	Few kernels with "dents" embryo growth
9.4	All kernels with "Dents", dry matter near maximum
9	Grains mature and drying.

After Hon way, J.J., 1963. Special Report no. 48. Iowa State University, USA.

Fig. 5: Growth stages of maize.

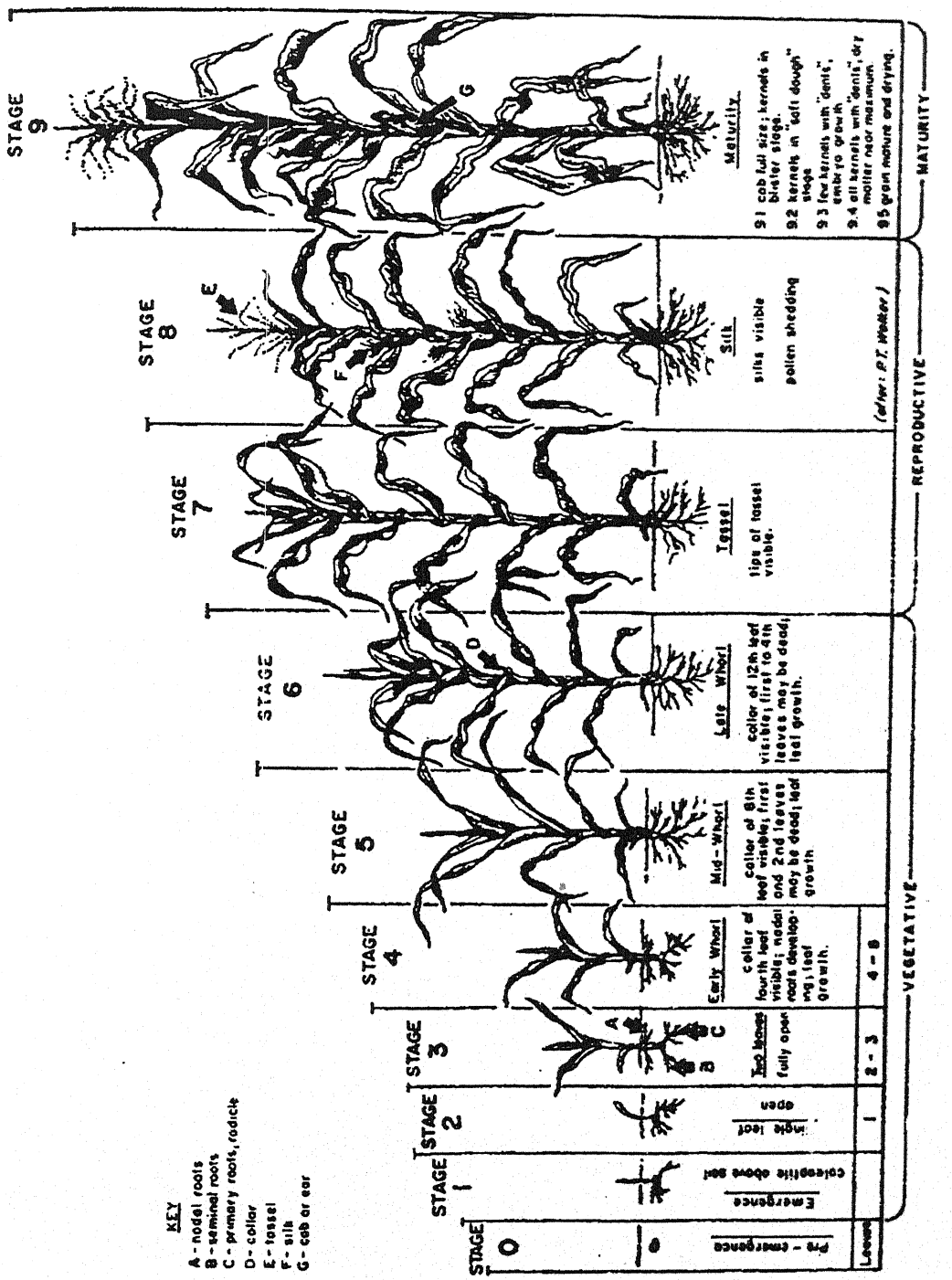


FIG. 5: GROWTH STAGES OF MAIZE.

4.0 Heavy infection, lesions abundant on lower and middle leaves, extending to upper leaves.

5.0 Very heavy infection lesions abundant on almost all leaves, plants prematurely dry or killed by the disease.

Using zero made a slight modification in the scale. This indicated no disease. The disease reaction was recorded on individual plant basis on 7th, 14th, 21st day after the inoculation. The disease reaction between the ratings 0-0.9 immune or near immune (I), 1.0 - 2.0 was considered as resistant (R), 2.1-2.5 as moderately resistant (MR), 2.6-3.0 as moderately susceptible (MS), 3.1-3.5 as susceptible (S) and > 3.6 as highly susceptible (HS).

3.12 Latent Period

The time between the inoculation of disease and the start of the production of spore by resulting infection called latent period. The observation of latent period was calculated as hours.

3.13 Disease Intensity

For the observation of disease intensity, average infected leaf per plant and average total leaf per plant of

each genotype were counted and disease intensity was calculated by apply following formula:

$$\text{Disease intensity} = \frac{\text{Average infected leaves/plant}}{\text{Average total leaves/plant}} \times 10$$

3.14 Sporulation

Number of spores produced in each spots or lesion called sporulation. Fresh and healthy spots cut into the plant and put with 1 ml distilled water in culture tube and shake. One drop of this spore suspension place on haemocytometer and count the spore numbers.

3.15 Spot size

Measurement of area occupied by lesion / spot known as spot size. For measuring spot size of infected leaves spot area were calculated by multiplying length and width of the spots and represented in millimeter.

3.16 Infection efficiency

Average proportion of deposited spores that produce functional lesions is called infection efficiency. For the observing infection efficiency, 1 ml spore suspension (100 spores/ml H_2O) was spread on healthy leaf. Plants were kept in moisture chamber for 24 h. After incubation period of 10 days plants were examined for lesions appeared on leaf.

3.17 Basic infection Rate

The basic infection rate is that measures any increases or decreases in the area under active sporulation (R). The R-value represents the rate of multiplication of the sporulating part of the disease symptom. The R was calculated by following formula:

$$R = \frac{1}{x_0(t_2 - t_1)}\left\{in\frac{1}{1-x_2} - in\frac{1}{1-x_1}\right\}$$

Where: x_1 and x_2 are disease severity scores

t_1 and t_2 = time

X_0 = Initial disease severity

3.18 AUDPC

Area Under Disease Progress Curve (AUDPC) was calculated by using the formula suggested by Wilcoxson *et al.* (1975).

$$AUDPC = \sum_{i-1}^{k} \tfrac{1}{2}(S_i + S_{i+1})\, d$$

3.19 Presentation of Data

Data were presented in graphics or tabular form. Data were statistically analyzed. Statistical analyses were made through computer programmer Excel 2000, Mstat C and Minitab 11 software.

RESULTS & DISCUSSION

Chapter- 4

RESULTS & DISCUSSIONS

4.1 Symptomology

Drechslera maydis, the causal agent of southern leaf blight (SLB) or southern corn leaf blight (SCLB), was serious in the Jhansi region. Infected tissue were extensively covered with spots and chlorosis rendering them non productive. *D. maydis* was found to have a higher saprophytic ability and hence high primary inoculum's levels are likely to be found in this area with high disease occurrence. *D. maydis* shows little or no specificity to plant genotypes and infect a wide range of plant genotypes.

The pathogen infects leaves, producing smaller lesions with parallel sides and little chlorosis. Spore production is influenced by temperature as more lesions were formed near 30°C than at low temperatures. The

large lesions observed in Jhansi and around are an indication of the host susceptibility and the prevailing weather conditions.

Matsumoto and Noguchi (1998) was studied influence of temperature on germination phase. On expression of symptom they reported that the disease incidence increased when the period of sowing was later and temperature was moderately low.

Pal and Kaiser (2001) reported that the plants are much vulnerable to the attack of disease when planting done in July, while early planting in May or June, or late planting in August reduced the incidence. Disease incidence gradually increased with the increase in plant density and it was maximum at a population of 70,000 ha-1 while it was minimum at 40,000 ha-1. Nitrogen alone or in combination with phosphorus and potassium, or with both phosphorus and potassium, reduced it. There was a gradual increase in the disease severity with the increase in the dose of nitrogen and the maximum infection occurred at the highest dose of nitrogen i e 160 kg ha-1. *In vitro* studies also showed that nitrogen significantly increased the linear growth of the pathogen, while both phosphorus and potassium individually or in combination with nitrogen reduced it. Nitrogen also significantly increased the percentage of conidial germination, while both phosphorus and

potassium individually or in combination with nitrogen reduced it.

Rai *et al.*, (2002) found a definite relation between environmental parameters and the disease development in maize. Disease developments were related with the age of the host as the first incidence was noticed at 55 days of plant growth. Chang and Hwang (2003) observed that leaf blight development gradually increased. Leaf blight severity was significantly higher on the lower (older) leaves then on the upper (younger) leaves. Inoculum's density increased from 103 to 106 conidia per ml, the development of leaf blight increased. Disease severity also increased as the time of leaf wetness duration increased from 0 to 60 h. Wetness during above 48 h and a high inoculum density (106 conidia per ml) caused severe leaf blight symptoms in adlay seedlings.

The symptoms were observed on diseased plants as lesions on leaves and were small dot like in early crop stage. After few days' spots become spindle like or elliptical in shape with yellow green colour or chlorotic about 2 to 9 mm long and 1 to 2 mm wide. On maturity spots turned light to dark brown in colour, targeted like zonate. The spots were parallel to leaf veins. Developed individual lesions occurred singly or coalesced with other to give a blighted appearance (Fig. 6). Extensive and sporulating lesions were observed on stem, flower bract and leaves on severely infected plants. Prior to this study

Fig.6: Disease symptoms,
Healthy leaves and infected leaf.

Tsukiboshi *et al.*, (1996) also observed similar symptoms. They reported that 75 isolate from maize produced only small and chlorotic lesions in the *rhm* resistant maize lines, 1 isolate caused severe symptoms with, typical and large lesions. The increased ability to cause lesion enlargement of the isolate in the *rhm* resistant line was attributed to the increase in the number of appressoria formed at the time of spore germination and faster hyphal extension in the host cells. Gong and Zhang (2002) observed fractal analysis of lesion patterns of SLB. Fractal characteristics of lesion patterns were described by using fractal geometry and fractal dimensions determined by perimeter area and curve length method. Fractal dimension of maize southern leaf blight were 1.1633.

Oikawa *et al.*, (2004) reported that when the causal agent of SCLB *Bipolaris maydis* was inoculated on the third leaf, the amount of 2-hydroxy-4, 7-dimethoxy -1, 4-benzoxzin-3-one glycoside (HDMBOA-GIC) increased, reaching a maximum level after 48h of inoculation. The infection of *B. maydis* induced formation of dark brown lesions, where Bx-related compound 6-methoxy-2-benzoxazolinone (MBoA) was most abundantly present. The latter is formed by degradation of DIMBoA and HDMBOA; the HDMBOA-GIC was most abundant in the surrounding green tissues. Among the Bx-related compounds, MBoA exhibited the strongest inhibition of

the germination of the conidia and growth of germ tubes of *B. maydis*.

4.2 Prevalence of SLB in Jhansi and adjoining areas.

Maydis leaf blight is common maize disease and widely spread in and around Jhansi district (Fig. 7). Regular surveys were conducted during Kharif season 2003-2005 at twenty eight places in Jhansi district *viz.* Hansari taparian, Bejoli, Pali pahadi, Raksha, Ambabai, Baruwasagar, Ghughua, Sakrar, Bangra, Mauranipur, Dhawaker, Dhamana payak, Revan, Namoni, Ghurat, Bada gaon, Chira gaon, Semry, Baral, Amara, Moth, Punch, Arach, Gursarai, Todi fatepur, Bhatta gaon, Marry and Buda bhojla. Twenty plants were randomly selected at each site to record the incidence and severity of disease. Maximum disease incidence and severity were recorded in Bangra, Mauranipur, Samry Villages while minimum was recorded at Ambabai (Table 10). Samples were collected from each place for further studies. The results are presented in table 11. Maximum conidial length (61.63 µm) was recorded in samples collected from village Marry while minimum conidial length (44.43 µm) was recorded in Bada gaon samples. Maximum width of conidia (14.52 µm) from Nemoni and minimum width of conidia (8.12 µm) from Bada gaon were recorded. Similarly, maximum number of septa (6.51) was found in conidia of Nemoni village and minimum number of septa (4.55) was found in conidia of Ambabai village.

Fig. 7: Geography of Jhansi district.

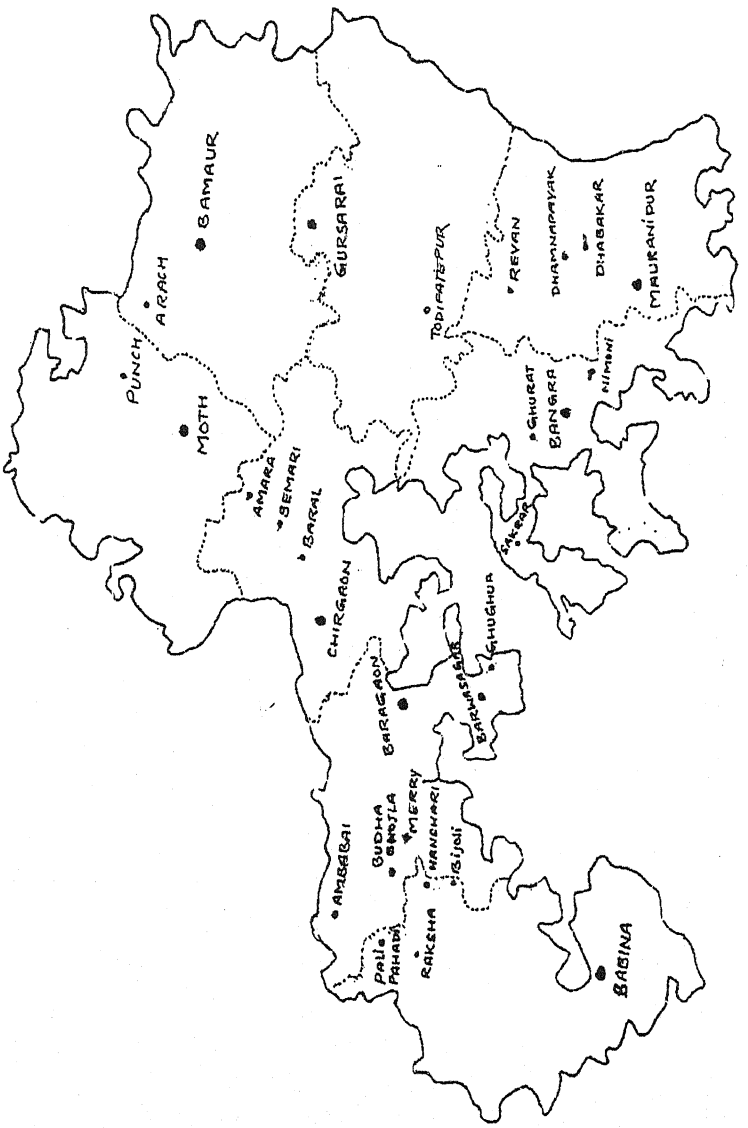


Fig. 7: Geography of Jhansi

Table 10: Incidence & severity of SLB of maize in various villages of Jhansi district

Place	No. of plant study	No. of Infected plants	Incidence (%)	Severity (%)
Hansari Taparian	20	17	85	15
Rajgadh	20	15	75	12
Pili Pahadi	20	11	55	10
Raksha	20	13	65	12
Ambabai	20	9	45	8
Barwasagar	20	16	80	15
Ghughua	20	17	85	16
Sakrar	20	16	80	14
Bangra	20	20	100	22
Mauranipur	20	20	100	23
Dhawaker	20	19	95	20
Dhamnapayak	20	17	85	18
Revan	20	18	90	19
Nimoni	20	16	80	17
Gurat	20	14	70	16
Badagaon	20	17	85	19
Chirgaon	20	19	95	18
Semry	20	20	100	22
Baral	20	17	85	16
Amara	20	16	80	13
Moth	20	13	65	14
Bhattagaon	20	19	95	18
Marry	20	18	90	16
Budha	20	15	75	15
Todifatepur	20	17	85	17
Gursarai	20	18	90	18
Arach	20	16	80	16
Punch	20	19	95	20

Table 11: Size of conidia belonging to pathogen of Various localities

Place	Length (μ)	Width (μ)	No. Of Septa / conidia
Hansari Taparian	52.55	9.31	5.51
Rajgadh	53.15	10.21	6.11
Pili Pahadi	55.47	12.61	4.92
Raksha	54.40	10.32	5.72
Ambabai	45.12	8.76	4.55
Barwasagar	56.67	14.12	6.61
Ghughua	57.43	13.45	6.31
Sakrar	50.63	10.56	5.76
Bangra	48.37	9.32	4.47
Mauranipur	53.34	11.12	5.01
Dhawaker	51.41	12.86	5.81
Dhamnapayak	50.21	13.56	5.10
Revan	52.11	11.22	5.92
Nimoni	56.62	14.52	6.51
Gurat	54.42	10.46	4.90
Badagaon	44.43	8.12	4.67
Chirgaon	47.72	9.92	5.94
Semry	46.92	10.13	5.78
Baral	49.12	8.71	4.99
Amara	51.62	11.47	5.23
Moth	53.73	13.21	6.14
Bhattagaon	56.38	10.28	5.64
Marry	61.63	13.43	6.39
Budha	60.16	12.11	5.53
Todi fate pur	51.11	10.77	4.91
Gursarai	55.52	11.32	5.42
Arach	49.17	9.40	5.11
Punch	51.19	12.61	4.74
Average	52.52	11.21	5.49
SD	4.10	1.76	0.61

* Average of 50 conidia for each isolate

Barba *et al.*, (2004) studied conidial morphology. Their studies also describe the effect of growing substrate on the size, septation and morphology of conidia, as well as the relationship of inoculum's density to disease intensity. The various substrates included six culture media, seeds and fresh leaves of barley, wheat (*Triticum aestivum*), rye (*Secale cereale*), and triticale (*Triticum secalotricum*). Conidia formed in culture media (68.2 x 21.9 µm; 5.7 pseudosepta) and on seeds (78. 3 x 20.4 µm; 7.2 pseudsepta) were shorter, wider, and with less septa than those from leaf lesions (92.9 x 18.2 µm; 7.7 pseudosepta). The effect of the inoculum density (ID) and disease intensity (DI) were tested by applying spore suspensions (2.5 x 10^3, 5.0 x 10^3, 10.0 x 10^3, 15.0 x 10^3, and 20.0 x 10^3 conidia/ml) to plants of the barley cultivars BR-2. The ID/DI relationship was represented by a quadratic model equation, in which the maximum values of 183 lesion/leaf and 79% disease severity were obtained with 16,500 and 14,000 conidia/ml, respectively. The number of conidia required for one leaf lesion was estimated in 50 to 90.

4.3 Isolation & Pathogenicity Test

The pathogen was isolated and multiplied on sorghum seeds. Inoculation was made on 10 days old seedlings and these were kept in glass house under controlled conditions. After 10 days, seedlings were

observed for disease appearance. The test confirmed the Koch's postulate.

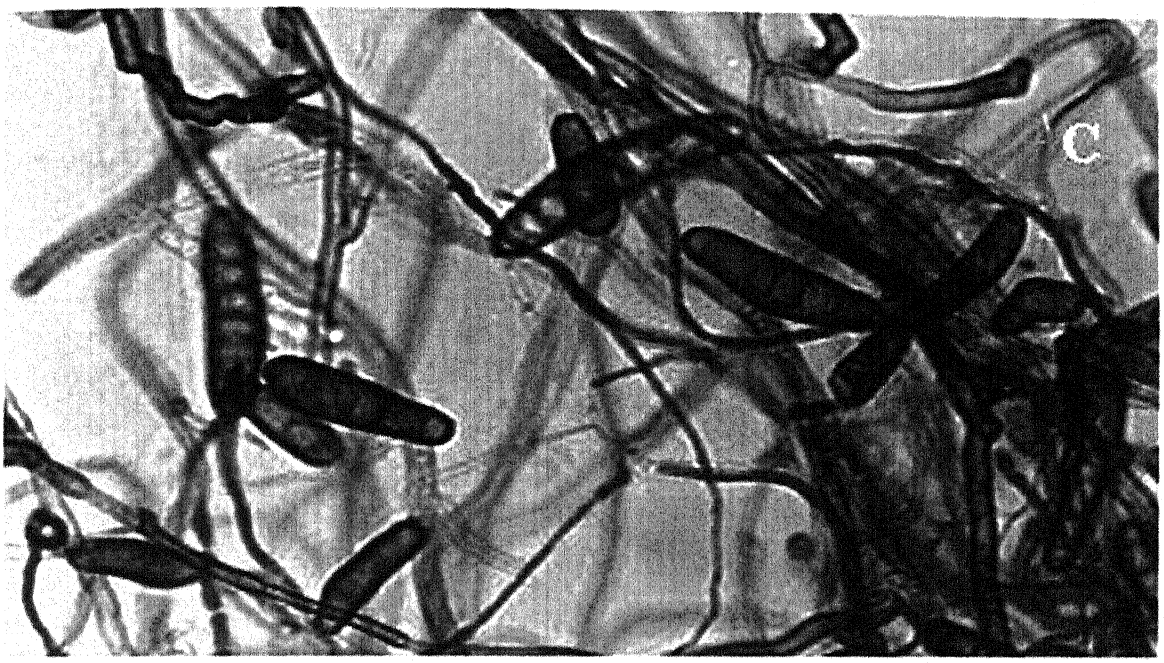
4.4 Morphological Cutural Character

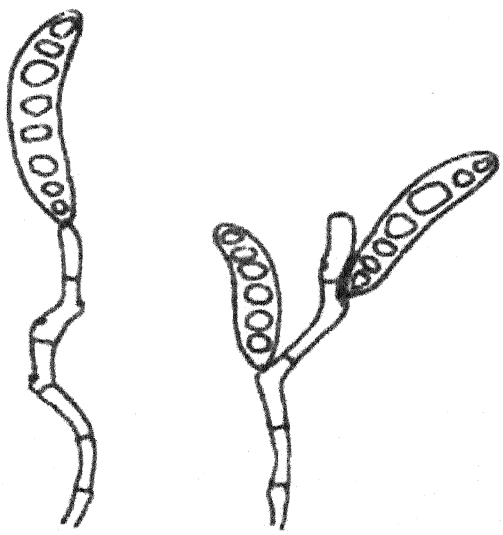
After isolation and multiplication of pathogen, a temporary mount was prepared in lecto-phenol and cotton blue for examining the morphological characteristics of the pathogen. On examining it reveals that colonies on PDA was effuse grey, blackish brown to dark black in colour. Conidiophores arising in grc up from flat dark brown stroma, straight to flexuous, some times geniculate, cicatrized, conidiogenous nodes smooth amber brown, pale near the apex, smooth up to 52.52 µ (44.4-61.6 µ) long, 11.2 µ (8.12-14.52 µ) in the broadest part, 5.49 µ (4.47- 6.61 µ) pseudoseptate. Hilum was thick, dark often flat, non-protruding in nature. Telomorphic state was not observed in natural field conditions (Fig. 8).

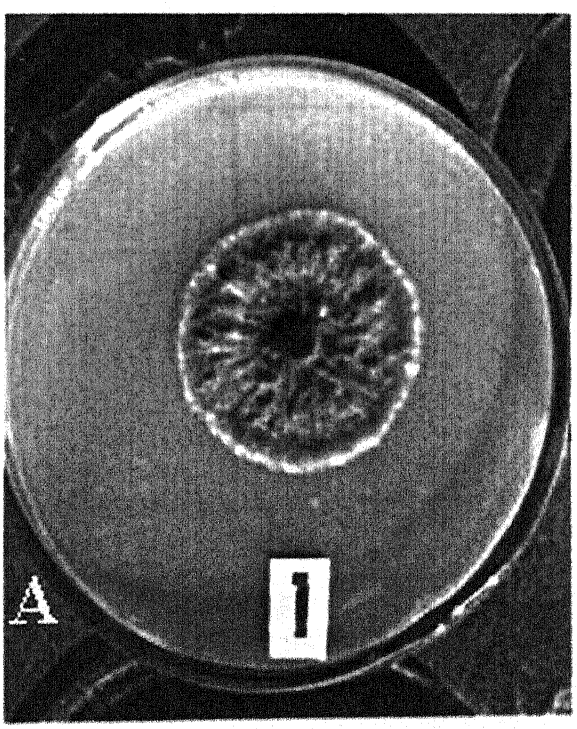
Germination behaviour of conidia under vari us incubation perio ds was also studied in moisture chamber at 30°C. The results are shown in table 12, 13and 14. Table 12 indicates the length of hyphae after incubating 25 to 50 hours. Results showed that hyphae increase in length with time duration. Minimum i.e. 33 µ length was measured after 25 hours of incubation. Maximum length of hyphae was recorded at 50 hours (925 µ).

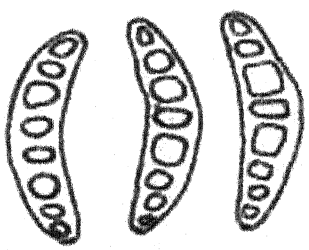
Fig.8: Conidia with conidiophores of *D. maydis.*

(A) Colony on Czapek dox medium
(B) Camera Lucida drawing
(C) Microphotograph









B

Table 12: Length of hyphae after incubation of 25 to 50 hr at 30° C

Time after inoculation (hr)	**Length of hyphae** (μ)
25	33
30	86
35	168
40	330
45	551
50	925

Table 13: Length of hyphae and number of hyphal cells composing the colonies 29, 44, 50 and 76 hr after inoculation*

Time after inoculation (hr)	**Length of hyphae (μ)**	**Number of hyphal cells**
29 **	160	3
44 ***	864	18
50 ****	1368	28
76 *****	7356	130

* Inoculation at 1 pm of September 22nd, 2004
** Observation at 6 pm of September 23rd, 2004
*** Observation at 9 am of September 24th, 2004
**** Observation at 3 pm of September 24th, 2004
***** Observation at 5 pm of September 25th, 2004

Table 13 indicates the length of hyphae and number of hyphal cells composing the colony after 29, 44, 50 and 76 hours. It is from the data that maximum hyphal length was observed at / after 76 h and (7356 µ) and maximum number of hyphal cells i.e. 130.

Table 14 indicates the length of axial hyphae twc to twelve days after incubation and daily difference were calculated between 201 µ to 378 µ.

Chen *et al.*, (2003) studied the temperature for spore germination of *B. sorghicola* and *B. sorokiniana* [*Cochliobolus sativus*] At 45°C the germination rates were 85.1% for *B. specifera* and 2.3% for *B. sorokiniana.* Nutrient solution, [*pH* was 6.47) including soil suspension, could stimulate the germination of *B. specifera,* after 4h and *B. sorghicola* after 2h spores needed water to germinate but were not sensitive to light.

Different characters viz. average total leaf/plant, average infected leaf/ plant, number of spots/leaf and spot size were also recorded in different maize genotypes under pot conditions. The results are shown in Table 15. Average and standard deviation of total leaf/plant were found 5.8 and 0.65, respectively. Highest average total leaf/plant was recorded 7.2 in genotype Sikkim collection GP-13 and lowest was 4.2 in IG01 - 534. Average infected leaf/plant of genotypes was calculated

Table 14: Length of axial hyphae 2 to 12 days after Inoculation at 30°C

Days after inoculation	Length of axial hyphae (μ)	Daily difference (μ)
2	176	-
3	377	201
4	690	313
5	1051	361
6	1369	318
7	1686	297
8	2028	362
9	2406	378
10	2664	258
11	2995	331
12	3278	283

Table 15: Average leaf, infected leaf per plant, number of spots & spot size after inoculation in pot conditions

S.N.	Genotype	Average total leaf/plant (1)	Average infected leaf/ plant (2)	Number of spots/leaf (3)	Spot size (mm) (4)
1	African tall	5.0	3.4	10.6	5.36 x 1.40
2	J-1006	5.4	4.0	39.2	3.68 x 1.32
3	IG01-769	5.6	4.8	24.8	4.02 x 1.42
4	IG01-532	5.8	5.2	18.2	4.96 x 1.42
5	IG01-534	4.2	4.0	17.4	3.38 x 1.08
6	Seti (local)	6.6	6.2	20.6	2.78 x 1.02
7	IG01-770	6.4	5.0	11.8	3.44 x 1.10
8	IG01-767	6.4	4.6	26.8	4.06 x 1.10
9	Sikkim collection GP-13	7.2	6.8	12.8	8.16 x 1.82
10	IG01-729	6.4	5.8	15.4	7.46 x 1.72
11	IG01-535	5.6	4.4	17.2	3.18 x 1.04
12	IG01-728	6.2	4.0	21.4	4.54 x 1.20
13	Paheli-1	5.2	4.0	38.6	2.90 x 1.06
14	IG01-746	5.4	3.8	41.8	3.16 x 1.06
15	IG01-788	5.2	4.2	33.0	2.58 x 1.0
16	IG01-674	7.0	5.2	29.6	3.16 x 1.26

17	IG01-575	6.2	4.8	40.8	2.52 x 1.02
18	IG01-804	6.0	4.4	32.6	3.88 x 1.14
19	IG01-691	5.6	4.0	31.2	7.72 x 2.04
20	IG01-471	6.4	5.0	43.6	4.66 x 1.44
21	IG01-792	6.8	3.0	51.4	3.12 x 1.04
22	IG01-750	5.2	4.0	21.0	4.98 x 1.38
23	IG01-799	6.0	3.6	20.4	3.64 x 1.16
24	IG01-790	5.2	3.6	25.0	3.30 x 1.00
25	IG01-711	5.4	4.6	30.2	3.40 x 1.10
26	IG01-807	5.4	4.8	18.8	3.20 x 1.04
27	IG01-678	5.4	4.2	14.6	2.66 x 1.04
28	IG01-806	6.0	4.2	17.0	3.5 x 1.12
29	IG01-782	6.0	4.8	25.8	4.14 x 1.2
30	IG01-709	6.0	4.8	36.0	3.9 x 1.14
	Average	**5.8**	**4.5**	**26.3**	**4.04x1.23**
	SD	**0.65**	**0.80**	**10.53**	**1.44x0.25**

4.5 with standard deviation 0.80. Highest average infected leaf/plant was recorded 6.8 in Sikkim collection GP-13 and lowest was recorded 3.0 in IG01-792. Average and standard deviation of number of spots/leaf in genotypes was calculated as 26.3 and 10.53, respectively. Highest number of spots/leaf was recorded 51.4 in IG01-792 and lowest 10.6 in African tall. Spot sizes were also measured in millimeter. Average spot sizes of thirty genotypes were calculated to be 4.04 x 1.23 mm (SD 1.44 x 0.25). Maximum length of spots was observed to be 8.16 mm in Sikkim collection GP-13 and minimum length of spots was observed 2.52mm (IG01-575). Maximum width of spots was as 2.04 mm and minimum width 1.0 mm was found in genotypes IG01-788, IG01 - 790 respectively.

Green *et al.*, (2004) observed disease severity increased with increasing leaf wetness at 15, 20, 25, 30 and 32°C. At 10°C, conidia of the fungi showed minimal germination, regardless of leaf wetness duration however, an increase in conidial germination, appressorium formation occurred at 15°C whereas the optimum temperature for appressorium formation of *D. gigantean* was 23°C. Maximum disease occurred after 48h of leaf wetness at 32°C for *D. gigantean.* Disease caused by the fungi decreased when 4h of continuous leaf wetness was followed by a 22h dry period.

4.5 Optimal growth requirement of the Pathogen

Fungal growth characters on different media have been summarized in table 16. The growth was highly variable with regard to colony diameter and colony characteristics. Maximum growth was observed on czapek's medium and minimum in malt extract. The pathogen growing on czapek's and corn meal agar medium produced dark greenish white and superficially well developed colonies with thicker mycelia. Whereas on malt extract medium and oatmeal agar medium produced light gray to black superficial colonies with thin mycelia. Colonies on Richard's medium and PDA were dark blackish white superficial poorly branched. Colonies on Czapek's dox, malt extract medium and oatmeal agar were deeply submerged /embedded (Fig. 9&9a).

Effect on growth of *D. maydis* of different culture media viz. Czapek's dox medium (CDA), Corn Meal Agar (CMA), Oat Meal Agar (OMA), Potato Dextrose Agar (PDA), Richard's medium, Malt Extract Agar (MEA) medium and temperature at 20°C, 25°C, 30°C, 35°C, 40°C is depicted in Fig. 10. The growth of colony showed an increasing trend with temperature but after 35°C it reduced significantly. The data indicated that over all maximum radial growth of 74.8 mm was observed in CDA at 35°C temperature and minimum radial growth of 7.0 mm in PDA at 40°C temperature. Individually

Table 16: Cultural characteristics of *D. maydis* in different media.

S. No.	Media	Colour	Growth		Remark
			Superficial	Submerged	
1.	Czapek's Dox	Dark greenish white	Well developed and branched	Deeply embedded	+++++
2.	Corn meal Agar	Dark greenish white	Well developed	Less developed	+++
3.	Oat meal Agar	Light Gray to black	Well developed Profusely Branched	Deeply embedded	+++++
4.	Potato Dextrose Agar	Dark Blackish white	Developed poorly branched	Less developed	++++
5.	Richard	Dark Blackish white	Developed poorly branched	Less developed	+++
6.	Malt Extract Agar	Light gray to black	Poorly branched	Deeply embedded	++

++ Poor
+++ Average
++++ Good
+++++ Excellent

Fig.9: Growth of *D. maydis* in different culture media (Graph).

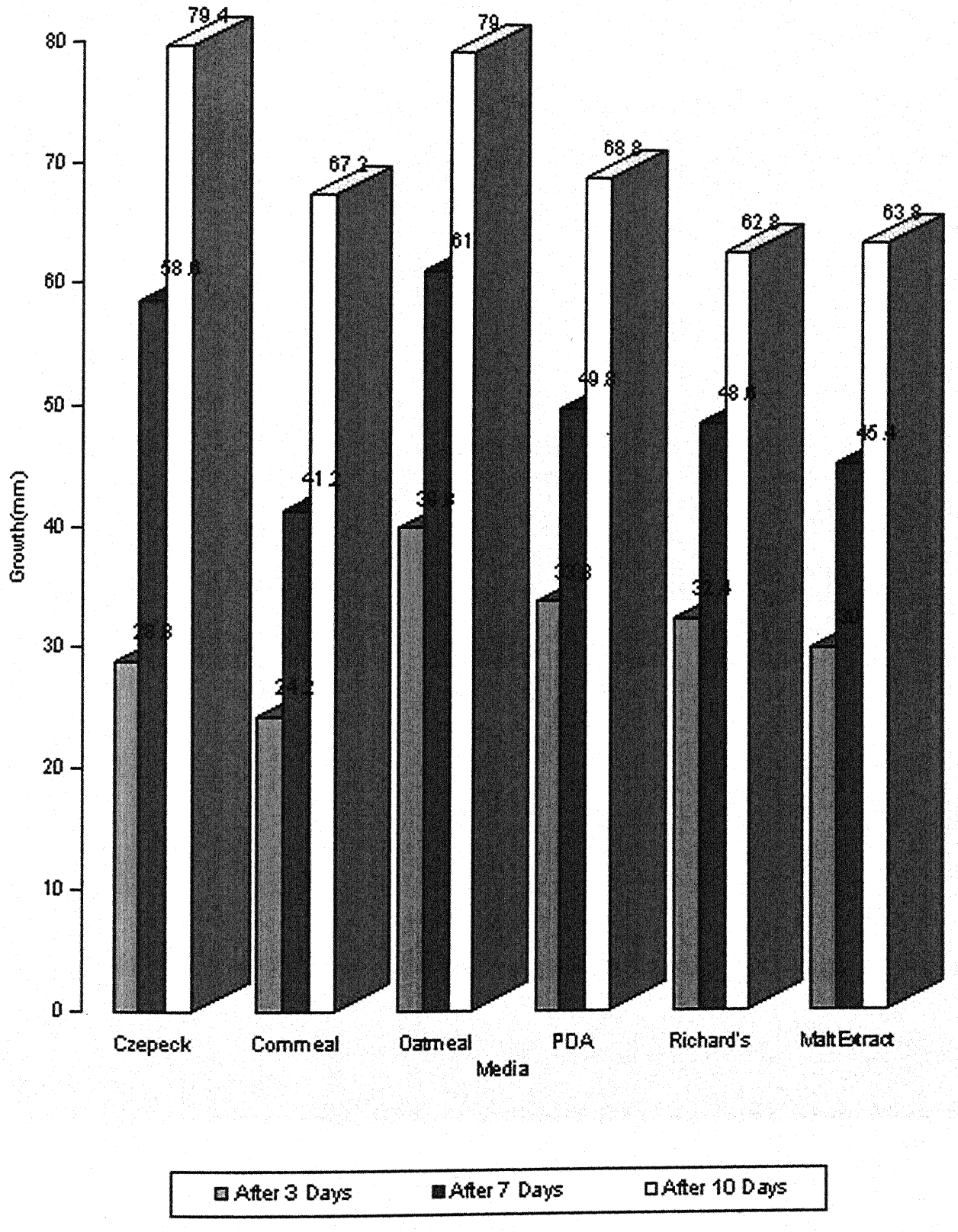


Fig.9 Growth of *D. maydis* in different media

Fig.9:(a) Growth of *D. maydis* in different culture media (Photo).

(1) Czepeck dox medium
(2) Corn meal agar medium
(3) Oat meal agar medium
(4) Potato dextrose agar medium
(5) Richard's medium
(6) Malt extract agar medium

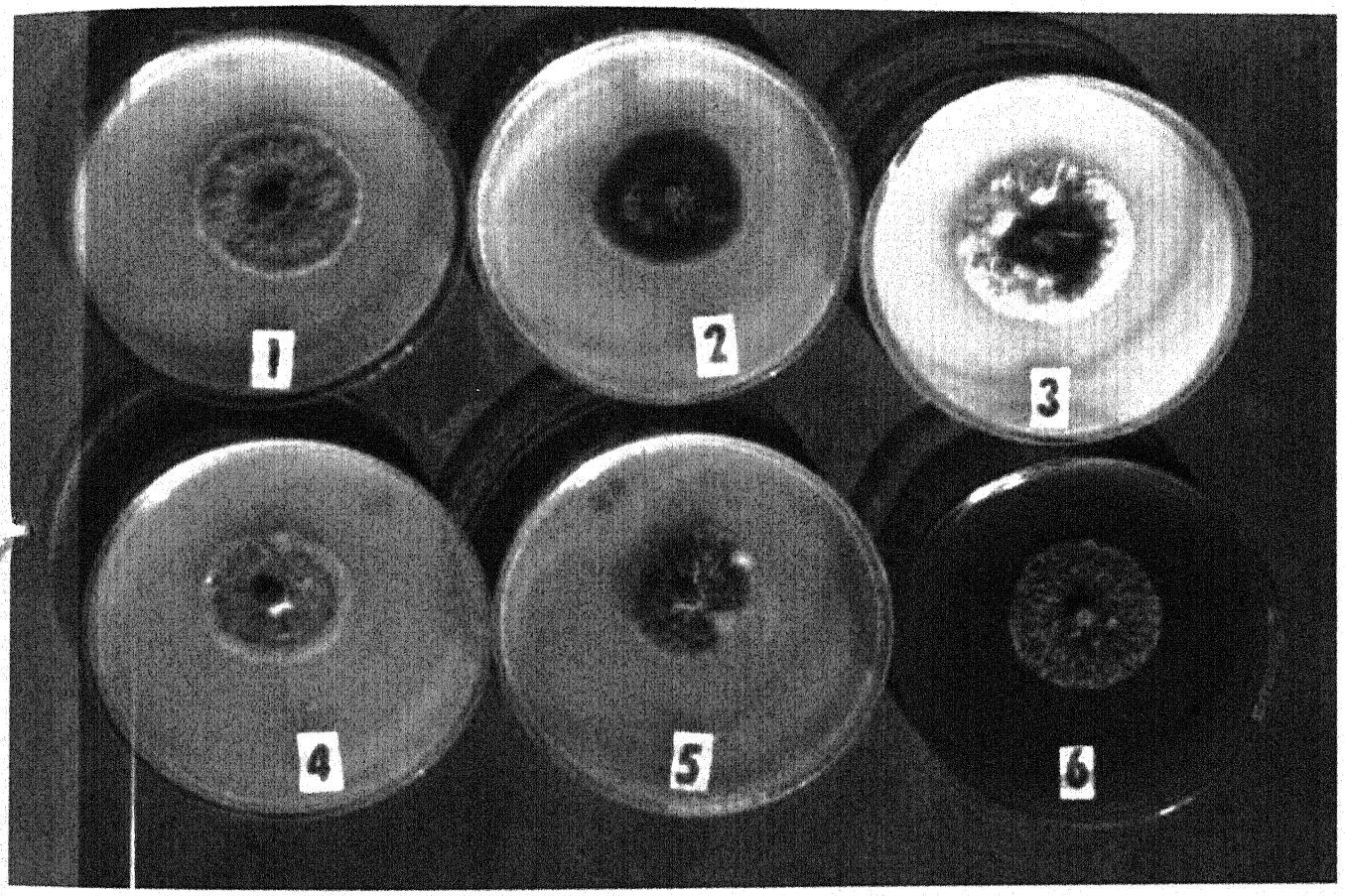

1
2
3
4
5
6

Fig.10: Effect of different temperature on growth of *D. maydis.*

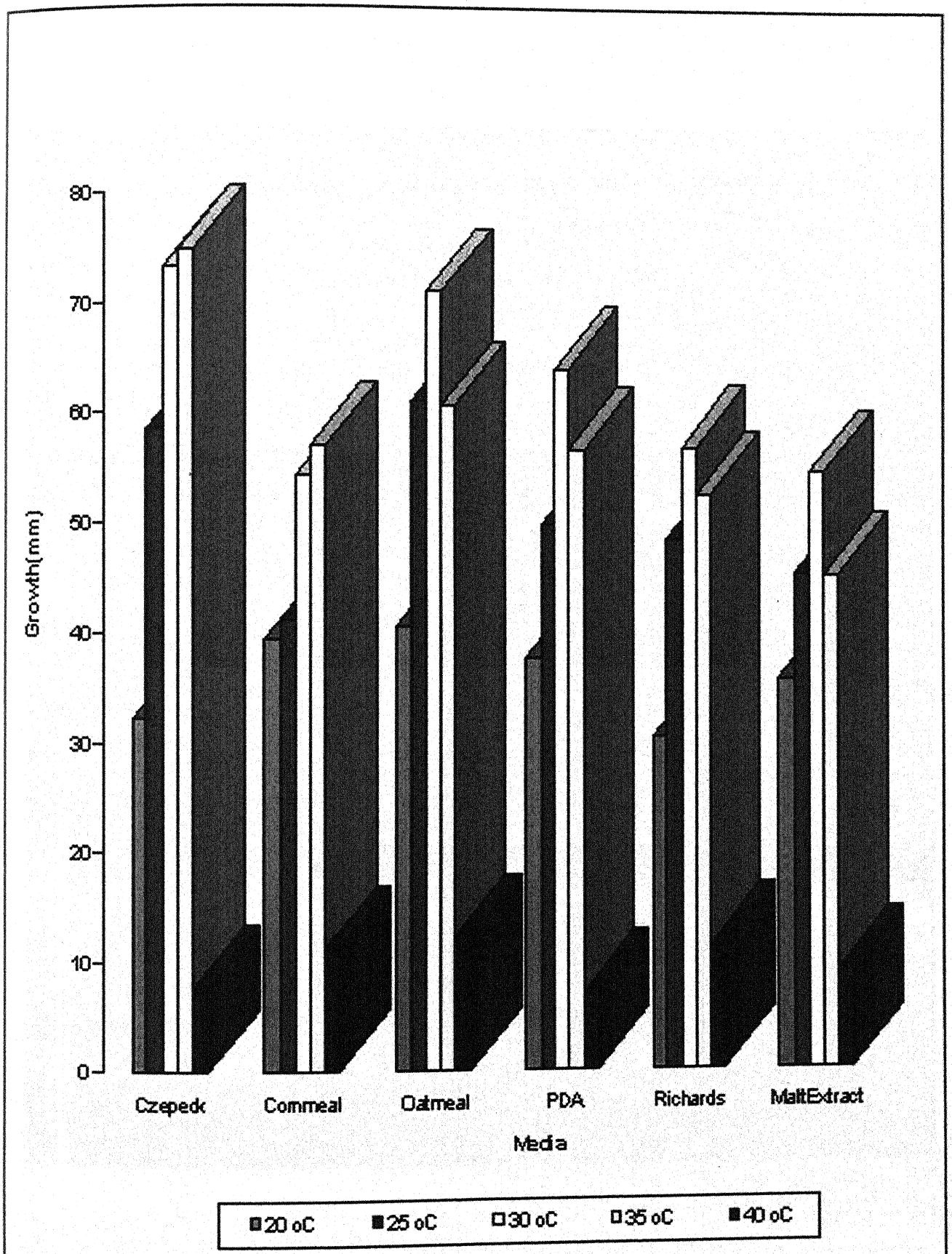


Fig.10 Effect of Temperature on growth of *D. maydis*

maximum radial growth in CDA medium was also observed as 74.8 mm at 35°C and minimum radial growth as 07.4 at 40°C. In CMA medium maximum radial growth of 57 mm was observed at 35°C and minimum growth of 11.3 mm at 40°C. In OMA medium 71.0 mm was observed as maximum radial growth at 30°C and minimum radial growth as 12.2 mm at 40°C. In PDA medium maximum radial growth was 64.0 mm at 30°C and minimum was 7.0 mm at 40°C. In Richard's medium maximum radial growth of 56.8 mm was observed at 30°C and minimum as 11.4 mm at 40°C. In malt extract agar medium maximum radial growth of 54.6 mm was observed at 30°C and minimum radial growth as 9.2 mm at 40°C.

At 20°C maximum radial growth of 40.6 mm was found in oatmeal agar medium and minimum of 30.6 mm in Richard's medium. At 25°C maximum radial growth of 61.0 mm was found in oatmeal agar and minimum as 41.2 mm in corn meal agar medium. At 30°C maximum radial growth of 73.2 mm was found in Czapek's dox medium and minimum as 54.2 mm in corn meal agar medium. Maximum radial growth of 74.8 mm was found in CDA medium and minimum of 45.2 mm in MEA medium at 35°C. Maximum radial growth of 12.2 mm was found in OMA and minimum as 7.0 mm in PDA medium at 40°C. Average maximum radial growth of 62.30 mm was observed at 30°C and minimum as 9.76

mm at 40°C. So 30°C is the effective temperature for fungal growth. On an average CDA medium and OMA medium were equally effective (49.28 and 49.08 respectively) and MEA showed poor radial growth (38.0 mm) at various temperature.

Ali *et al.*, (1992) also found the similar trends. They reported that out of five different incubation temperatures (10, 20, 25, 30 and 35°C) the highest radial colony growth of fungus (87 mm) was recorded at 30 degree centigrade. Among the three culture media, maize leaf agar proved to be the best culture substrate in showing the maximum colony growth (79.83 mm) and spore size (74 x 13 mm) of the fungus. Pal and Kaiser (2003) reported that the mycelial growth and conidial germination of the pathogen *Drechslera maydis* Nisikado race '0' were influenced by temperature. The optimum temperature was being 33°C. Relative humidity did not have any impact on mycelial growth and conidial germination. The situation was true on the disease incidence recorded in the field. Linear growth was highest at 33°C in petri plates, and minimum at 21°C and 38°C below and above 33°C, mycelial growth gradually decreased. Mycelial growth was good at 70-100% RH, but was optimum at 90-100%. Below 90% RH, colony growth was slow, conidial germination was also highest at 33°C. Germination percentage was high between 27 and 32°C, but gradually decreased below

27°C and above 33°C. *In vitro* studies were also indicated that both temperature and RH affected linear growth and conidial germination. During the experimentation (August and September 1985), the daily mean maximum temperature was 32.5°C and 32.0°C, the daily mean maximum RH was 96.2% and 97.0% and the total monthly rainfall was 347.0 and 437.5 mm, respectively. Although the mean maximum temperature was higher (32.9 - 33.1°C) in September, the temperature was less (31 - 32°C) in August during 1994 and 1996 compared to 1995.

4.6 Perpetuation of the pathogen

However, in case of SLB of maize few studies have successfully been completed for the annual recurrence (over winter) of the disease through survival of pathogen in form of mycelium (dormant) or air borne nature of conidia from distant localities. In very few cases the seed infection has been found to be a primary source of inoculum, particularly in race 'T'. In case of present investigation, the observations revealed that the role of infected soil and seed are not possible in perpetuation of the disease as plants grown in infected soil and or plants raised from seeds collected from infected plants did not show any disease symptoms. Survival of inoculum's / dormant mycelium in plant debris has been observed in TTC test and it was also confirmed by inoculating maize seedlings by infected leaf powder (fig. 11). So it can be

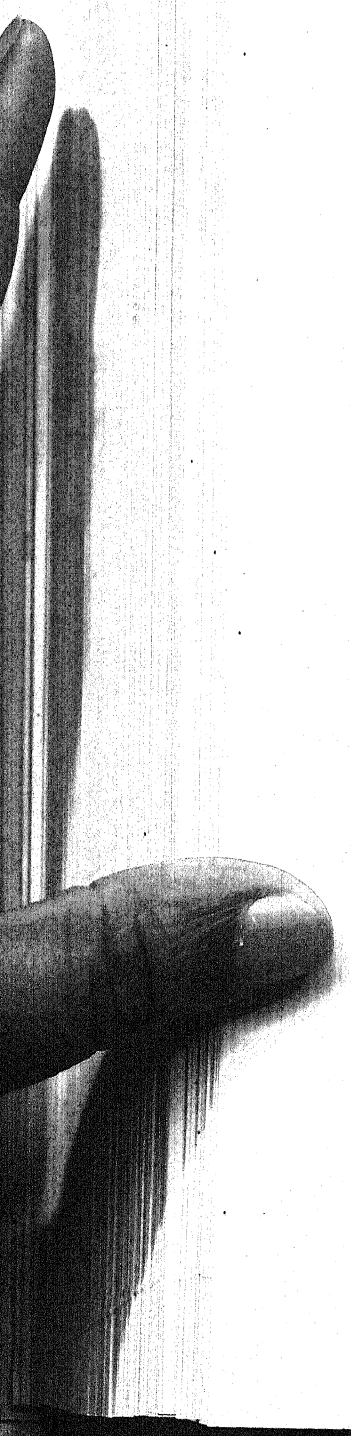
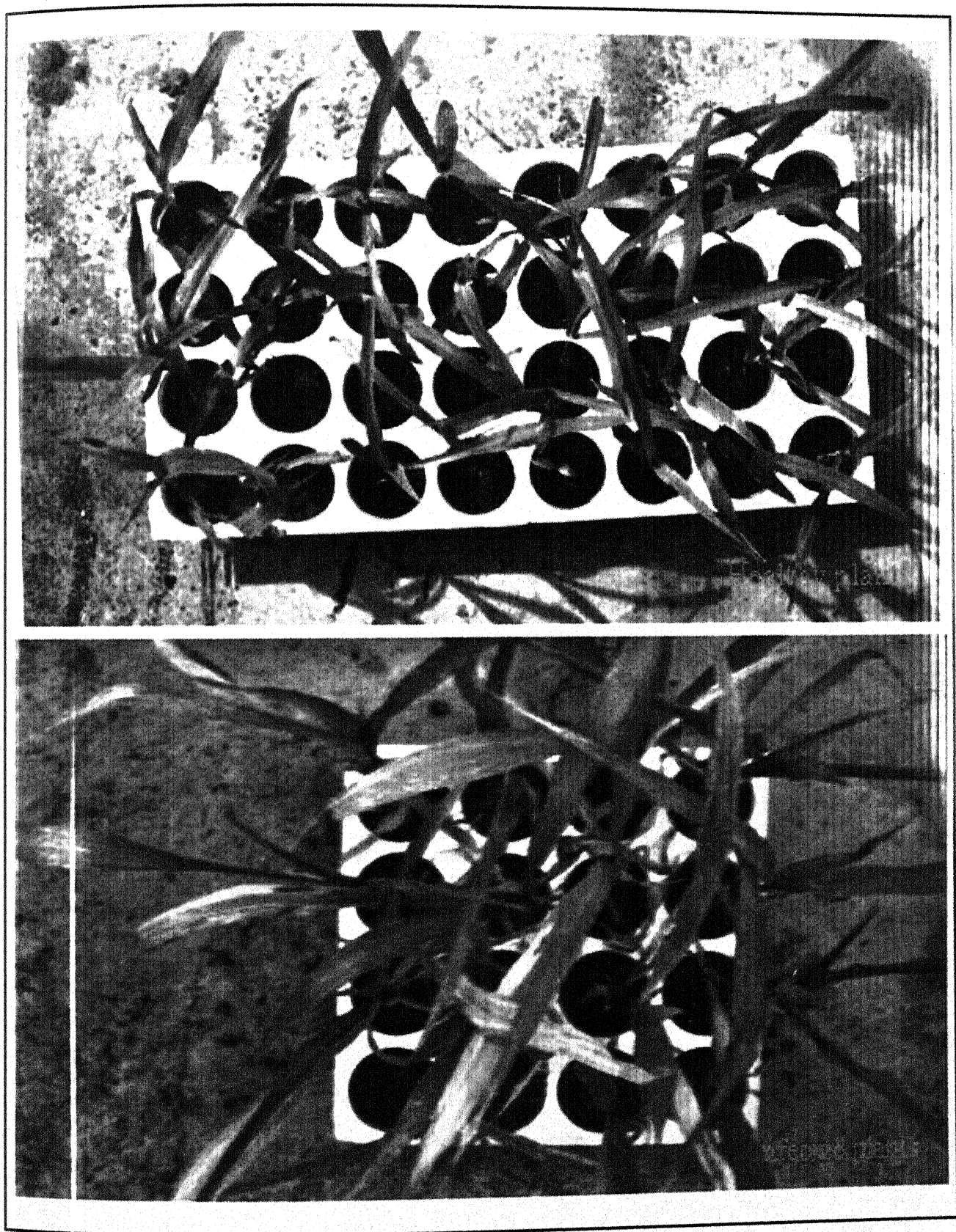


Fig.11: Pathogenicity test of *D. maydis* in maize seedlings.



concluded that plant debris plays a positive role in bringing about infection in next crop season. Possibility of secondary spread through air was also established by experiments. There are also possibilities of other infected host plants serving as a source of primary inoculum in the off-season. However, it requires testing of large number of host species for collateral host.

4.7 Screening of Maize Genotype

Thirty-genotype were selected for analysis for resistance against southern leaf blight disease. The passport data of these genotypes is summarized in table17.

Maize genotypes were screened in controlled conditions and results are given in table 18. All the thirty maize genotypes are divided into seven categories (Reaction type) viz. Immune (I), Highly Resistant (HR) Resistant (R), Moderately Resistant (MR), Moderately Susceptible (MS) Susceptible (S) and Highly susceptible (HS) on the 0-5 point scale. None of the genotypes was found highly resistant against maydis leaf blight. Genotype African tall was found resistant. Another genotype J-1006 was found moderately resistant. Nine genotypes viz. IG01-535, IG01-728, IG01-674, IG01-804, IG01-792, IG01-799, IG01-678, IG01-806, and IG01-782 were found moderately susceptible. Maximum number of genotype were found susceptible *viz.* IG01-769, IG01-

Table 17: Passport data of selected maize genotypes

S. No.	Accession	IG No.	Pedigree	Origin
1	African tall	-	Released variety	Mexico
2	J – 1006	-	Released variety	Indigenous
3	Hyd 997-1758	IG01 – 769	CUBA 126	6327 (Mex)
4	-	IG01 – 532	-	-
5	-	IG01 – 534	-	-
6	Seti (Local)	-	Collection	NEH India
7	Hyd. 997-1762	IG01 – 770	TRIN GP1	6331 (Mex)
8	Hyd.997 - 1750	IG01 – 767	CUBA 113	6319 (Mex)
9	GP - 13	-	Collection	Sikkim
10	Hyd 997 - 1666	IG01 – 729	CUBA T-1-48	6235 (Mex)
11	-	IG01 – 535	-	-
12	Hyd 997 - 1665	IG01 – 728	CUBA T-1-44	6234 (Mex)
13	Paheli - 1	-	Collection	NEH India
14	Hyd 997-1688	IG01 – 746	CUBA T -82	6257 (Mex)
15	Hyd 997-1528	IG01 – 788	SNLP 105	6097 (Mex)
16	Hyd 997-1517	IG01 – 674	02 POOL 32 C16	6086 (Mex)
17	Hyd 997-1518	IG01 – 675	02 POOL 33 C23	6087 (Mex)
18	Hyd 997 -1551	IG01 – 804	VERA 207	6120 (Mex)
19	Hyd 997 - 1571	IG01 – 691	BRAZ 1177	6140 (Mex)
20	-	IG01 – 471	-	-

21	Hyd 997 – 1532	IG01 – 792	VERA 190	6101 (Mex)
22	Hyd 997 – 1700	IG01 – 750	OAXA 821	6269 (Mex)
23	Hyd 997 - 1544	IG01 – 799	SNLP 99	6113 (Mex)
24	Hyd 997 -1530	IG01 – 790	VERA 213	6099 (Mex)
25	Hyd 997 - 1634	IG01 – 711	BRAZ SE0 28	6203 (Mex)
26	Hyd 997 - 1556	IG01 – 807	BRAZ 928	6125 (Mex)
27	Hyd 997 - 1521	IG01 – 678	POBLAC 63 C2	6090 (Mex)
28	Hyd 997 - 1554	IG01 – 806	BRAZ 881	6123 (Mex)
29	Hyd 997 - 1508	IG01 – 782	ARZM0 1114	6077 (Mex)
30	Hyd 997 - 1627	IG01 – 709	CMS 06	6196 (Mex)

Table 18: Reaction of various genotypes against SLB of maize

S.N.	Reaction Type	No. of Genotype	Genotype
1	Immune (I)	0	None
2	Highly Resistant (HR)	0	None
3	Resistant (R)	1	African tall
4	Moderately Resistant (MR)	1	J-1006
5	Moderately Susceptible (MS)	9	IG01-535, IG01-728, IG01-674, IG01-804, IG01-792, IG01-799, IG01-678, IG01-806, IG01-782
6	Susceptible (S)	16	IG01-769, IG01-532, IG01-770, IG01-767, Sikkim Collection GP-13, IG01-729, Paheli-1, IG01-746, IG01-788, IG01-575, IG01-471, IG01-750, IG01-790, IG01-711, IG01-807, IG01-709
7	Highly Susceptible (HS)	3	IG01-534, Seti local, IG01-691

532, IG01-770, IG01-767, Sikkim collection GP-13, IG01-729, Pahel-1, IG01-746, IG01-788, IG01-575, IG01-471, IG01-750, IG01-790, IG01-711, IG01-807 and IG01-709. But only three genotype *viz* IG01-534, Seti local and IG01-691 were found to be highly susceptible.

AUDPC was also calculated. According to A- value genotypes are ranked into eighteen groups and presented in table 19. Minimum A-value 94.50 was calculated at rank one in genotype African tall and maximum A-value 157.50 was calculated at rank eighteen in genotype IG01 -534. Rank 1, 2, 4, 5, 7, 9, 11, 16 and 18 have only one genotype each. Rank 3, 6, 8, 10, 13, and 17 have two genotypes each. Rank twelve; fourteen and fifteen include three genotypes in each group. Here it is imperative to mention that rank one (African Tall) is comparatively slower blighting variety than other groups. Group 18 was fast blighting to SCLB.

4.8 Assessment and categorization of various attributes of Maize - SLB patho-system

Table 20 shows the various attributes (mono or polycyclic) of Maize - SLB patho-system in different maize genotypes. These attributes are Field reaction (F), Latent period (L), Spot size (SS), Diseases intensity (D), Sporulation (S), Infection efficiency (I), Basic infection rate (B) and Area under disease progress curve (A). The data indicates that maximum field reaction (4.00) was

Table 19: The rank of various genotypes against SLB of Maize according to area under disease progress curve.

Rank	"A" Value	No. of Genotype	Genotype
1	94.50	1	African tall
2	115.50	1	IG01-806
3	120.75	2	IG01-674, IG01-799
4	122.50	1	IG01-792
5	124.25	1	IG01-728
6	126.00	2	IG01-535, IG01-782
7	127.75	1	IG01-804
8	129.50	2	Sikkim Collection GP-13, IG01-678
9	131.25	1	IG01-807
10	133.00	2	IG01-770, IG01-767
11	134.75	1	IG01-709
12	138.25	3	J-1006, IG01-746, IG01-750
13	140.00	2	IG01-769, IG01-532
14	141.75	3	IG01-788, IG01-382, IG01-711
15	143.50	3	IG01-729, IG01-575, IG01-790
16	145.25	1	Paheli-1
17	148.75	2	Seti local, IG01-691
18	157.50	1	IG01-534

Table 20: Monocyclic / Polycyclic characters governing disease epidemic in selected maize genotypes.

S. No.	Genotype	Field reaction (F)	Latent period (hrs) (L)	Spot size (mm) (SS)	Disease Intensity (D)	Sporulation (Spores/ lesion) (S)	Infection efficiency (lesions/ 1000 spores) (I)	Basic infection rate unit/ day (B)	AUDPC (A)	Reaction type
	1	2	3	4	5	6	7	8	9	10
1	African tall	2.28	94	11.9	75	5,600	12.2	0.0105	94.5	R
2	J-1006	3.21	81	4.9	73	15,300	35.4	0.0157	138.25	MR
3	IG01-769	3.35	72	6.3	86	8,450	23.7	0.0070	140.00	S
4	IG01-532	3.35	69	12.4	91	9,550	20.0	0.0070	140.00	S
5	IG01-534	3.71	65	3.7	96	16,660	17.2	0.0070	157.50	HS
6	Seti local	4.00	45	3.1	92	12,655	21.9	0.0106	148.75	HS
7	IG01-770	3.21	66	3.8	85	6,450	14.2	0.0070	133.00	S
8	IG01-767	3.14	69	4.4	77	7,725	26.2	0.0084	133.00	S
9	Sikkim collection GP-13	3.14	68	16.9	95	6,552	17.5	0.0169	129.50	S
10	IG01-729	3.42	73	14.6	92	10,125	20.4	0.0070	143.50	S
11	IG01-535	3.00	78	3.4	76	7,255	17.8	0.0000	126.00	MS
12	IG01-728	2.92	82	9.8	70	5,844	23.0	0.0157	124.25	MS
13	Paheli-1	3.42	70	3.1	84	11,330	33.5	0.0035	145.25	S
14	IG01-746	3.35	68	3.6	79	10,675	39.1	0.0106	138.25	S
15	IG01-788	3.35	69	2.8	83	8,255	30.2	0.0035	141.75	S
16	IG01-674	2.85	84	4.4	80	6,466	29.8	0.0042	120.75	MS
17	IG01-575	3.42	75	2.7	84	11,450	37.0	0.0070	143.50	S
18	IG01-804	3.07	77	4.4	76	6,225	33.1	0.0035	127.75	MS

19	IG01-691	3.57	62	15.0	80	8,566	30.2	0.0030	148.75	HS
20	IG01-471	3.35	70	6.4	78	7,725	40.7	0.0035	141.75	S
21	IG01-792	2.92	83	3.8	64	6,000	43.5	0.0084	122.50	MS
22	IG01-750	3.28	72	13.8	82	7,550	21.0	0.0035	138.25	S
23	IG01-799	2.85	88	7.8	76	6,250	24.4	0.01570	120.75	MS
24	IG01-790	3.42	66	4.1	81	6,566	26.2	0.0001	143.50	S
25	IG01-711	3.35	69	4.7	91	12,750	28.8	0.0035	141.75	S
26	IG01-807	3.14	74	4.2	90	8,750	20.3	0.0035	131.25	S
27	IG01-678	3.07	79	3.4	88	6,890	17.9	0.0210	129.50	MS
28	IG01-806	2.71	94	4.7	73	6,025	19.8	0.0105	115.50	MS
29	IG01-782	3.00	78	5.4	82	7,000	26.1	0.0000	126.00	MS
30	IG01-709	3.14	72	4.9	84	11,000	35.2	0.0157	134.75	S
	Av	**3.20**	**73.73**	**6.48**	**82.1**	**8721.3**	**26.21**	**0.0078**	**133.99**	
	SD	**0.32**	**9.77**	**4.21**	**7.68**	**2880.95**	**8.22**	**0.0055**	**12.30**	

observed in Seti local and minimum (2.28) in African tall. Average field reaction was 3.20. Maximum latent period (94 h) was noted in African tall and minimum (45 h) in Seti local. Average latent period was 73.73 h with standard deviation 9.77 h. Maximum spot size was noticed 6.48 mm in Sikkim collection GP-13 and minimum spot size was 2.7 mm in IG01-575 (Av = 6.48). Average disease intensity 82.1 with standard deviation 7.68 was calculated. Maximum disease intensity (96) was found in IG01-534 and minimum (64) in IG01-792 was recorded. Spores per lesion (Sporulation) were also counted; results revealed that maximum sporulation (16,660 spores per lesion) in IG01-534 and minimum (5600) in African tall with an average of 8721.3 with standard deviation 2880.95. Average infection efficiency (lesion /1000 spores) was calculated as 26.21 with standard deviation of 8.22. Maximum infection efficiency of 43.5 was recorded in IG01-792 and minimum (12.2) in African tall. Average Basic infection rate was calculated to be 0.0078 with standard deviation of 0.0055. Maximum basic infection rate i.e. 0.0210 was found in IG01-678 and minimum (0.001) in three genotypes *viz.* IG01-535, IG01-790 and IG01-782. Area under disease progress curve was maximum (157.50) in IG01-534 minimum 94.5 in African tall, was recorded with an average 133.99 and standard deviation 12.30.

Maize crop in all areas of India was found to have a heavy pathogen inoculum's load that undermined the leaf area and weakened the plants resulting in very poor crop stands. Favourable environmental conditions operating on poor disease resistance genome generated epidemics involving *E. turcicum, P. maydis, B. maydis, P. sorghi* and *P. polysora,* ideally, lowland rust is favoured by 16 hours of free moisture on the plant for germination of uredospores (Hollier and King, 1985).

Results also indicated that ideal temperature and moisture conditions coupled with susceptible genotypes could result in yield losses.

After evaluations of Vgandan maize germplasm for resistance to *E. turcicum,* Adipala *et al.,* (1993b) reported that all had necrotic susceptible reactions when inoculated with races 0, 1, 23 and 23N, but for the observed resistance had no resemblance to the known *Ht* gene expression.

While the Indian fodder maize improvement program has done a tremendous job in producing maize varieties for the different ecological zones, it is evident that the aspect of disease control by genetic resistance must be allotted more attention. This is because all the recommended varieties were found to be very susceptible to two or more of the pathogens observed in the fields. It was interesting to observe that cultivar African tall was

resistant. Pataky (1994) reported that high levels of partial resistance with or without *Ht*-genes presented a spectacular approach in reducing damage from NLB on sweet corn which also eliminates the severe yield depressing chlorosis associated with *Ht* gene resistance in very susceptible backgrounds. Unfortunately no reports were available in case of SLB. Studies by Carson (1995b) indicated that latent period is related to partial resistance which suggests that selection for increased latent period length would be more beneficial than selecting for reduced disease severity. Selection for increased latent period length can be done in environments without severe disease epidemics, and also breeding material could be assessed as seedling for latent period length in the greenhouse during the off-season. Levy (1991) showed that isolates from different areas were of different parasitic fitness as indicated by infection efficiency, sporulation and spot size, while isolates of the same location showed less variation. Inoculum of the previous crop has been found to be critical in epidemic build up for subsequent cropping especially in non-tillage systems, as reported by Pedersen and Oldham (1992) using race 2. While non-tillage is not a common practice in India, the heavy inoculum's production means a lot of primary inoculum in subsequent plantings. According to Gevers (1975) the *Ht N* major gene of resistance derived from the Mexican maize variety Pepitrla is reasonably stable, but in some

parts of world the effects may fail to be expressed and genetic segregation may not behave like expected of dominant genes ratios, but does however remain in the tolerable limits of deviation of stability and segregation. He suggested the occurrence of biotypes in India able to overcome *Ht N* gene resistance. The *Ht N* gene is also background sensitive, and high temperature reduced symptom expression on line B37 *Ht* 3. The plants were evaluated at 26°C day 22°C nights and 22°C day/ 18°C night temperatures. There was observable weakened virulence at high temperatures. Combining *Ht* 1 and *Ht* 3 genes did not result in significantly less disease from those homoxygons for each gene (Leath and Pederson, 1986).

Spot size is one of the components of susceptibility of a host to a fungus disease (popular 1978). Hamid *et al,* (1982) used spot length to estimate the resistance of several- maize inbreeds lines to different isolates of race 3 of *B. zeicola.* We examined field symptom expression as an attempt to gauge levels of resistance in the field as could be indicated by lesion sizes and complexes of the various diseases. They present for the first time record of extremely large spots of over 6 cm by 0.5 cm for *B. maydis.* Njuguna *et al.*, (1992) reported spot size for *E. turcicum* to be between 5 to 10 cm long and 1.3 cm wide in Kenya. Most of the lesions were observed in the upper canopy zones of the plants and hence may adversely

affect yield. Pataky (1992) found that yield losses were significant when disease incidence was severe and present on the upper canopy. Their data agree with that of Levy and Leonard (1990). Serious yield depression is therefore expected because most of the lesions were found in the upper third of the canopy. The critical population of leaves needed by the plants for dry matter production is in the upper canopy and is related to the final yield (Pataky 1992). Raymundo (1978) and Solomonovich (1992) found that when the lower third of all the leaves were removed, no yield loss was observed. There is a clear indication that most of the fodder maize varieties grown in India are very susceptible to *D. maydis* inducing the observed disease in this study. Many of the lesions were sporulation, which is an indication of susceptibility (Hooker 1961). While extensive chlorosis was seen on some hybrids infected with *D. maydis*. The Jhansi environmental conditions were very conducive to disease development and therefore we suggest genotype evaluation for resistance is done under severe inoculum pressure and favorable environment. The locations with high rainfall high humidity had severed disease and more lesions, which were sporulation. Screening of genotypes should be done for more than one disease per ecological zone, as it is the standard practice for maize breeders.

According to Tharmmasak *et al*, (1995) the inoculum potential and inoculum effectiveness of *Helminthosporium turcicum* was positively and beneficially; enhance the screening for disease resistance of NCLB program. Medium compositions, inoculums concentration, plant age and inoculation technique were included in this experiment. The best medium to support maximum sporulation was sterile seed of Higari sorghum ($20x10^5$ conidia/ml). The effective inoculums concentration was $1x10^5$conidia/ml at all plant ages.

Carson (1998) reported saprophytic or over wintering phase of the life cycle of *Cochliobolus heterostrophus,* the causal organism of southern leaf blight of maize may be a factor in the persistence of apparently less aggressive isolates in the pathogen population. Carson (1998) studied the aggressiveness and percent perennation (over wintering survival) of *C. heterostrophus*. He found significant negative correlation between the ability of isolates to persist on the soil surface and their aggressiveness. The ability of race '0' isolates to sporulate on senescent corn leaf discs was positively correlated with their aggressiveness selection against aggressiveness during over wintering does not appear sufficient by itself to counter selection for increased aggressiveness occurring during the pathogen's pathogenic phase.

Dhanju and Sain (2005) identified and involved the set of stable lines of maize in the development of stable disease resistant hybrids.

Shivankar and Shivankar (2000) studied that the plants inoculated with spore suspension of *H. turcicum* at 45 days after sowing showed leaf light incidence was higher in inoculated plants.

Cai *et al.*, (2003) applied a combination of amplified fragment length polymorphism (AFLP) technique and bulked segregant analysis (BSA) to a large F2 population in order to identify molecular markers linked to the *rhm* gene for resistance to SCLB. The marker useful for map-based cloning for the *rhm* gene and marker assisted selection for *rhm*.

Sharma *et al.*, (2003) studied that incorporation of maydis resistance from two sources have been rather discouraging. The capacity of two sources to transmit their resistant gene though possessed high *per se* resistance, have not been very fruitful under different background germplasm. To understand the reason behind such behaviour, genetic study based on combining ability was undertaken using two sources. Resistance to maydis leaf blight was predominantly under the influence of additive gene action along with significant contributions from additive x additive epistasis. However, significant role of dominant gene

action along with epistasis could not be ruled out entirely.

Carson *et al.*, (2004) found a total of 11 quantitative trait loci (QTLs) were responsible to condition resistance for SLB, depending upon which disease rating was used in the analysis. When the AUDPC data were combined and analyzed over environments, seven QTLs, on chromosomes 1, 2, 3, 4, 7 and 10 were found to come from the resistant parent Mo17. An additional QTL for resistance on chromosome 1 come from the susceptible parent B73. The eight identified QTLs accounted for 46% of the phenotypic variation for resistance QTL x environment interactions often were highly significant but, with one exception, were the result of difference in the magnitude of QTL effects between years and not due to changes in direction of effects. QTLs on the long arm of chromosome 1 and chromosomes 2 and 3 had the largest effects.

4.9 Contingency Analysis

Various attributes of maize - SLB patho-system belonging to different maize genotypes were coded for further analysis. All the characters were encoded in three groups i.e. Low (I) Medium (II) and High (III). Only one character (i.e. field reaction) was divided into four categories, the code was as I II III IV. The code III & IV belongs to high category and the remaining character *viz.*

Latent period, spot size, disease intensity, sporulation, infection efficiency, basic infection rate and area under disease progress curve were divided into three categories. The codes for various attributes are given in table 21. Encoded data was given in table 22.

After encoding of data, a contingency table was prepared and shown in table 23 to find out the relationship between Latent period (L), Spot size (SS), Disease intensity (D), Sporulation (S), Infection efficiency (I), Basic infection rate (B) and Area under disease progress curve (A) verses field reaction (F) in 3x4 matrices.

The tests of independence (Chi square test) were applied to find out the relationship of each character with field reaction. Results were shown in table 24, 25, 26, 27, 28, 29 and 30. Relationship between latent period and field reaction were shown in table 24. The data indicated that moderate or higher latent period have lower field reaction. Twenty-nine genotypes have moderate to higher latent period. Statistical analysis showed that chi-square (χ^2) calculated 21.96 are greater than chi-square (χ^2) tabulated value (*i.e.* 12.59 at 5%). So the null hypothesis was rejected and concluded that both the variables are dependent to each other. Contingency coefficient was significant (C=0.65).

Table 21: Codes for various characters

S. No.	Code	Field reaction	Latent Period	Spot size	Disease intensity	Sporulation	Infection efficiency	Basic Infection Rate	AUDPC
1	I (Low)	2.88 –2.71	42-60	2.7 – 7.4	64 –74	5600 – 9287	12.2 – 22.6	0.000 – 0.0070	94.5 – 115.5
2	II (Medium)	2.72 –3.14	67-78	7.5 – 12.2	75-85	9288 -- 12974	22.7 – 33.1	0.0071 – 0.00140	15.6 – 136.5
3	III (High)	3.15 –3.57	79-96	12.3 –17.0	86-96	12975 - 16661	33.2 – 43.6	0.0141 – 0.0210	136.6 – 157.5
4	IV (Highest)	3.58 –4.00	-	-	-	-	-	-	-

Table 22: Encoded data of Maize genotypes

S. No.	F	L	SS	D	S	I	B	A
1	I	III	II	II	I	I	II	I
2	III	III	I	I	III	III	III	II
3	III	II	I	III	I	II	I	III
4	III	II	III	III	II	I	I	III
5	IV	II	I	III	III	I	I	III
6	IV	I	I	III	II	I	II	III
7	III	II	I	II	I	I	I	II
8	II	II	I	II	I	II	II	II
9	II	II	III	III	I	I	III	II
10	III	II	III	III	II	I	I	III
11	II	II	I	II	I	I	I	II
12	II	III	II	I	I	II	III	II
13	III	II	I	II	II	III	I	III
14	III	II	I	II	II	III	II	III
15	III	II	I	II	I	II	I	III
16	II	III	I	II	I	II	I	II
17	III	II	I	II	II	III	I	III
18	II	II	I	II	I	II	I	II
19	III	II	III	II	I	II	I	III

20	III	II	I	II	I	III	I	III
21	II	III	I	I	I	III	II	II
22	III	II	III	II	I	I	I	III
23	II	III	II	II	I	II	III	II
24	III	II	I	II	I	II	I	III
25	III	II	I	III	II	II	I	III
26	II	II	I	III	I	I	I	II
27	II	III	I	III	I	I	III	II
28	II	III	I	I	I	I	II	I
29	II	II	I	II	I	II	I	II
30	II	II	I	II	II	III	III	II

I = Low **II = Medium** **III & IV = High**

Table 23: Contingency Table

	F_1	F_2	F_3	F_4
L_l	0	0	0	1
L_m	0	7	13	1
L_h	2	5	1	0
SS_l	1	9	10	2
SS_m	1	2	0	0
SS_h	0	1	4	0
D_l	1	2	1	0
D_m	1	7	9	0
D_h	0	3	4	2
S_l	2	11	7	0
S_m	0	1	6	1
S_h	0	0	1	1
I_l	2	5	4	2
I_m	0	6	5	0
I_h	0	1	5	0
B_l	0	5	12	1
B_m	2	2	1	1
B_h	0	5	1	0
A_l	2	0	0	0
A_m	0	12	2	0
A_h	0	0	12	2

Table 24: Test of Independence between field reaction and latent period

	F_1	F_2	F_3	F_4
L_l	0 (0.06)	0 (0.4)	0 (0.46)	1 (0.06)
L_m	0 (1.4)	7 (8.4)	13 (9.8)	1 (1.4)
L_h	2 (0.53)	5 (3.2)	1 (3.73)	0 (0.53)
χ^2 Calculated				**21.96**
χ^2 tabulated at 5%				**12.592**
Contingency coefficient (C)				**0.65**

Parentheses are expected values

HYPOTHESIS:

H_0 = both the variables are independent.

H_1 = both the variables are dependent.

STATISTICAL INFERENCE:

χ^2 (Calculated) $\geq \chi^2$ (Tabulated)

H_0 rejected

RESULT: field Reaction is dependent on Latent Period.

Relationship between spot size and field reaction were shown in table 25. The data indicated that Lower spot size have higher field reaction as twenty two genotypes belongs to this group while moderate and higher spot size have lower field reaction as three and five genotypes respectively belong to these groups. Statistical showed that chi-square (χ^2) calculated 69.64 are greater than chi-square (χ^2) tabulated value (i.e. 12.59 at 5%). So the null hypothesis was rejected and concluded that both the variables are dependent to each other. Contingency coefficient was also highly significant (C = 0.84).

Relationship between disease intensity and field reaction is presented in table 26. The data indicated that lower disease intensity have lower field reaction as only four genotypes belong of this group. While moderate disease intensity have maximum field reaction as seventeen genotypes belong of this group. Statistical inference was that chi square (χ^2) calculated 8.00 is less than chi-square (χ^2) tabulated value (i.e. 12.59 at 5%). So the null hypothesis was not rejected and concluded that both the variables are independent to each other. Contingency coefficient was moderately significant (C = 0.46).

Relationship between field reaction and sporulation is given in table 27. According to data medium to higher

Table 25: Test of independence between field reaction and spot size

	F_1	F_2	F_3	F_4
SS_l	1 (1.47)	9 (8.8)	10 (10.27)	11 (1.47)
SS_m	1 (0.20)	2 (1.20)	0 (1.4)	0 (0.20)
SS_h	0 (0.33)	1 (2.00)	4 (2.33)	0 (0.33)
χ^2 Calculated				**69.64**
χ^2 Tabulated at 5%				**12.592**
Contingency coefficient (C)				**0.84**

Parentheses are expected value

HYPOTHESIS:

H_0 = both the variables are independent.

H_1 = both the variables are dependent.

STATISTICAL INFERENCE:

χ^2 (Calculated) $\geq$ χ^2 (Tabulated)

H_0 rejected

RESULT: field Reaction is dependent on spot size.

Table 26: Test of independence between field reaction and disease intensity

	F_1	F_2	F_3	F_4
D_l	1 (0.27)	2 (1.60)	1 (1.90)	0 (0.27)
D_m	1 (1.13)	7 (6.8)	9 (7.93)	0 (0.27)
D_h	0 (0.60)	3 (3.6)	4 (4.2)	2 (0.60)
χ^2 Calculated				**8.00**
χ^2 Tabulated at 5%				**12.592**
Contingency coefficient (C)				**0.46**

Parentheses are expected value

HYPOTHESIS:

H_0 = both the variables are independent.

H_1 = both the variables are dependent.

STATISTICAL INFERENCE:

χ^2 (Calculated) $\leq \chi^2$ Tabulated)

H_1 rejected

RESULT: Field Reaction is independent on disease intensity.

Table 27: Test of independence between field reaction and Sporulation

	F_1	F_2	F_3	F_4
S_l	2 (1.33)	11 (8.00)	7 (9.33)	0 (1.33)
S_m	0 (0.53)	1 (3.2)	6 (3.73)	1 (0.53)
S_h	0 (0.13)	0 (0.8)	1 (0.93)	1 (0.13)
χ^2 Calculated				**13.99**
χ^2 Tabulated at 5%				**12.592**
Contingency coefficient (C)				**0.56**

Parentheses are expected value

HYPOTHESIS:

H_0 = both the variables are in dependent.

H_1 = both the variables are dependent.

STATISTICAL INFERENCE:

χ^2 (Calculated) $\geq$ χ^2 (Tabulated)

H_0 rejected

RESULT: Field Reaction is dependent on sporulation.

sporulation have higher field reaction as twenty and two genotype belong of these group. Statistical inference was calculated chi-square (χ^2) 13.96 are greater than chi square tabulated value (i.e. 12.59 at 5%). So the null hypothesis was rejected and concluded that both the variables are dependent to each other. Contingency coefficient was (C) 0.56.

Relationship between field reaction and infection efficiency is tabulated in table 28. The data indicated that lower infection efficiency have lower field reaction as thirteen genotypes belong to this group. Statistical inference calculated chi - square (χ^2) 9.05 is less than chi-square tabulated value (i.e. 12.59 at 5%). So the null hypothesis was accepted and concluded that both the variables are independent to each other. Contingency coefficient was (C) 0.48.

Relationship between field reaction and basic infection rate is shown in table 29, the data indicated that maximum genotypes have moderate basic infection rate and field reaction. Lower basic infection rate have higher field reaction as eighteen genotypes belong to this group. Moderate and higher basic infection rate have higher field reaction. Statistical inference was calculated chi-square (χ^2) 16.73 are greater than tabulated chi-square value (i.e. 12.592 at 5%). So the null hypothesis was rejected and concluded that both the variable are

Table 28: Test of in dependence between field reaction and Infection efficiency.

	F_1	F_2	F_3	F_4
I_l	2 (0.87)	5 (5.20)	4 (6.07)	2 (0.87)
I_m	0 (0.73)	6 (4.40)	5 (5.13)	0 (0.73)
I_h	0 (0.40)	1 (2.40)	5 (2.80)	0 (0.40)
χ^2 Calculated				**9.05**
χ^2 Tabulated at 5%				**12.592**
Contingency coefficient (C)				**0.48**

Parentheses are expected value

HYPOTHESIS:

H_0 = both the variables are independent.

H_1 = both the variables are dependent.

STATISTICAL INFERENCE:

χ^2 (Calculated) $\leq \chi^2$ (Tabulated)

H_1 rejected

RESULT: Field Reaction is independent on Infection efficiency.

Table 29: Test of in dependence between reaction and Basic infection rate.

	F_1	F_2	F_3	F_4
B_l	0 (1.2)	5 (7.2)	12 (8.4)	1 (1.2)
B_m	2 (0.4)	2 (2.4)	1 (2.8)	1 (0.4)
B_h	0 (0.4)	5 (2.4)	1 (2.8)	0 (0.4)
χ^2 Calculated				**16.73**
χ^2 Tabulated at 5%				**12.592**
Contingency coefficient (C)				**0.60**

Parentheses are expected value

HYPOTHESIS:

H_0 = both the variables are independent.

H_1 = both the variables are dependent.

STATISTICAL INFERENCE:

χ^2 (Calculated) $\geq \chi^2$ (Tabulated)

H_0 rejected

RESULT: Field Reaction is dependent on basic infection rate.

dependent to each other. Contingency coefficient (C) was 0.60.

Relationship between field reaction and area under disease progress curve is presented in table 30. The data indicated that lower area under disease progress curve have lower field reaction as only two genotypes belong this group. Higher area under disease progress curve has moderate to high field reaction. Statistical inference was calculated chi-square is greater than chi-square tabulated value. So the null hypothesis was rejected and concluded that both the variables are dependent to each other. Contingency coefficient was highly significant (C=0.80).

4.10 Correspondence Analysis

Correspondence analysis is a way to interpret relationship between row and columns by using chi-square technique. Chi-square analysis of various traits corresponding to field reaction and contribution of each trait of SLB epidemic is given in table 31. It is clear from the table that all the attributes contribute towards the field reaction. As Chi-square value of 74.39 (calculated) was high from critical chi-square value (tabulated) i.e. 61.51 at 5% significance level with 42 degree of freedom. The calculated statistic is greater than critical value so we rejected the null hypothesis and concluded that field

Table 30: Test of independence between field reaction and area under disease progress curve (AUDPC).

	F_1	F_2	F_3	F_4
A_l	2 (0.13)	0 (0.8)	0 (0.93)	0 (0.13)
A_m	0 (0.93)	12 (5.6)	2 (6.53)	0 (0.93)
A_h	0 (0.93)	0 (5.6)	12 (6.53)	2 (0.93)
χ^2 Calculated				**53.40**
χ^2 Tabulated at 5%				**12.592**
Contingency coefficient (C)				**0.80**

Parentheses are expected value

HYPOTHESIS:

H_0 = both the variables are independent.

H_1 = both the variables are dependent.

STATISTICAL INFERENCE:

χ^2 (Calculated) $\geq$ χ^2 Tabulated)

H_0 rejected

RESULT: Field Reaction is dependent on AUDPC.

Table 31: Chi-Square analysis of various traits of maize blight (SLB) epidemics

	F_1	F_2	F_3	F_4	Total
I_l	1.285	0.044	0.495	1.285	3.109
I_m	0.775	0.393	0.008	0.775	1.951
I_h	0.423	0.930	2.163	0.423	3.939
L_l	0.070	0.423	0.437	12.270	13.200
L_m	1.479	0.395	1.601	0.155	3.63
L_h	3.663	0.776	1.779	0.563	6.781
S_l	0.248	0.769	0.344	1.408	2.769
S_m	0.563	1.676	1.799	0.338	4.376
S_h	0.141	0.845	0.018	5.241	6.245
D_l	1.832	0.057	0.319	0.282	2.490
D_m	0.212	2.688	2.184	0.634	5.718
D_h	0.634	0.169	0.001	2.945	3.749
B_l	1.268	0.893	2.182	0.056	4.399
B_m	5.889	0.113	1.001	0.789	7.792
B_h	0.423	2.396	1.001	0.423	4.243
Total	18.905	12.567	15.332	27.587	χ^2 **=74.391**
					C= 0.844

reaction was dependent on various attributes of patho-system.

Row and column profiles of each attribute are presented in table 32 and 33. A brief examination of these tables confirms the dependence of field reaction on different attributes of patho-system as established in previous investigations.

In the terminology of correspondence analysis, the row and column totals of the matrix of relative frequencies are called the row mass and column mass, respectively. The mass profile of different parameters/ attributes was given in table 34. When examine the row wise, maximum mass i.e. 0.148 was occupied by medium latent period followed by low sporulation (i.e. 0.141) and low basic infection rate (0.127). Column wise, it was high in F3 (i.e. 0.437) followed by F2 (0.423). F1 and F4 occupy the same mass.

The term inertia in correspondence analysis is used by analogy with definition in applied mathematics of "moment of inertia" which stands for the integral mass time of the square distance to centroid. The relative inertias of different attributes are shown in table 35. It is evident from the table that maximum inertia (relative) occupied by low latent period (0.117) followed by medium basic infection rate (0.105) and high latent period (0.091). When observe column wise, maximum inertia

Table 32: Analysis of Row Profile (χ^2 %).

	F_1	F_2	F_3	F_4	Total
I_l	41.33%	1.42%	15.92%	41.33%	100%
I_m	39.72%	20.14%	0.41%	39.72%	100%
I_h	10.74%	23.61%	54.91%	10.74%	100%
L_l	0.53%	3.20%	3.31%	92.95%	100%
L_m	40.74%	10.88%	44.10%	4.27%	100%
L_h	54.02%	11.44%	26.24%	8.30%	100%
S_l	8.96%	27.77%	12.42%	50.85%	100%
S_m	12.87%	38.30%	41.11%	7.72%	100%
S_h	2.26%	13.53%	0.29%	83.92%	100%
D_l	73.57%	2.29%	12.81%	11.33%	100%
D_m	3.71%	47.01%	38.20%	11.08%	100%
D_h	16.91%	4.51%	0.03%	78.55%	100%
B_l	28.82%	20.30%	49.60%	1.27%	100%
B_m	75.58%	1.45%	12.85%	10.12%	100%
B_h	9.97%	56.47%	23.59%	9.97%	100%

Table 33: Analysis of Column Profile (χ^2 %).

	F_1	F_2	F_3	F_4
I_l	6.80%	0.35%	3.23%	4.66%
I_m	4.10%	3.13%	0.05%	2.81%
I_h	2.24%	7.40%	14.11%	1.53%
L_l	0.37%	3.37%	2.85%	44.48%
L_m	7.82%	3.14%	10.44%	0.56%
L_h	19.38%	6.17%	11.60%	2.04%
S_l	1.31%	6.12%	2.24%	5.10%
S_m	2.98%	13.34%	11.73%	1.23%
S_h	0.75%	6.72%	0.12%	19.00%
D_l	9.69%	0.45%	2.08%	1.02%
D_m	1.21%	21.39%	14.24%	2.30%
D_h	3.35%	1.34%	0.01%	10.68%
B_l	6.71%	7.11%	14.23%	0.20%
B_m	31.15%	0.70%	6.53%	2.86%
B_h	2.24%	19.07%	6.53%	1.53%
Total	100%	100%	100%	100%

Table 34: Mass profile of various attributes

	F_1	F_2	F_3	F_4	Mass (Row)
I_l	0.154	0.385	0.308	0.154	**0.092**
I_m	0.000	0.545	0.455	0.000	**0.077**
I_h	0.000	0.167	0.833	0.000	**0.042**
L_l	0.000	0.000	0.000	1.000	**0.007**
L_m	0.000	0.333	0.619	0.048	**0.148**
L_h	0.250	0.625	0.125	0.000	**0.056**
S_l	0.100	0.550	0.350	0.000	**0.141**
S_m	0.000	0.125	0.750	0.125	**0.056**
S_h	0.000	0.000	0.500	0.500	**0.014**
D_l	0.250	0.500	0.250	0.000	**0.028**
D_m	0.111	0.778	0.111	0.000	**0.063**
D_h	0.000	0.333	0.444	0.222	**0.063**
B_l	0.000	0.278	0.667	0.056	**0.127**
B_m	0.333	0.333	0.167	0.167	**0.042**
B_h	0.000	0.833	0.167	0.000	**0.042**
Mass (Columns)	**0.070**	**0.423**	**0.437**	**0.070**	**Total = 0.998**

Table 35: Relative Inertias of various attributes.

	F_1	F_2	F_3	F_4	Total (Row)
I_l	0.017	0.001	0.007	0.017	**0.042**
I_m	0.010	0.005	0.000	0.010	**0.026**
I_h	0.006	0.012	0.029	0.006	**0.053**
L_l	0.001	0.006	0.006	0.165	**0.117**
L_m	0.020	0.005	0.022	0.002	**0.049**
L_h	0.049	0.010	0.024	0.008	**0.091**
S_l	0.003	0.010	0.005	0.019	**0.037**
S_m	0.008	0.023	0.024	0.005	**0.059**
S_h	0.002	0.011	0.000	0.070	**0.084**
D_l	0.025	0.001	0.004	0.004	**0.033**
D_m	0.003	0.036	0.029	0.009	**0.077**
D_h	0.009	0.002	0.000	0.040	**0.050**
B_l	0.017	0.012	0.029	0.001	**0.059**
B_m	0.079	0.002	0.013	0.011	**0.105**
B_h	0.006	0.032	0.013	0.006	**0.057**
Total (Column)	**0.254**	**0.169**	**0.206**	**0.371**	**Total = 1.000**

was seen in F4 column. Where it was 0.371 followed by F1, F3 and F2.

The mass, inertia and contribution to axis of rows and columns are given in table 36 and 37. Analysis of contingency is given in table 38 indicates that most of the attributes placed near axis one. Its proportion is about 50 per cent (0.4999). Thirty eight per cent occupy the place near axis two and 0.1201per cent placed near axis three. Maximum inertia (χ^2) 0.2619 was associated with axis one followed by 0.1991 and 0.0629 with axis two and three, respectively.

4.11 Correlation Studies

Correlation studies of various attributes were also made. The results are presented in table 39. It is evident from the table that latent period and sporulation are negatively correlated with field reaction while area under disease progress curve (AUDPC) is significantly positively correlated with field reaction. Similarly, latent period has significant negative correlation with disease intensity and AUDPC.

Table 36: Correspondence analysis mass, inertia and contributions to axis (Row contributions)

S.N.	Name	Qual	Mass	Inert	Axis 1			Axis 2			Axis 3		
					Coord*	Corr**	Contr***	Coord	Corr	Contr	Coord	Corr	Contr
1	Il	1.000	0.092	0.042	-0.048	0.010	0.001	-0.476	0.947	0.104	0.102	0.043	0.015
2	Im	1.000	0.077	0.026	-0.097	0.053	0.003	0.349	0.686	0.047	-0.215	0.261	0.057
3	Ih	1.000	0.042	0.053	0.476	0.346	0.037	0.570	0.494	0.069	0.324	0.160	0.071
4	Ll	1.000	0.007	0.177	1.993	0.301	0.107	-2.890	0.633	0.295	-0.934	0.066	0.098
5	Lm	1.000	0.148	0.049	0.284	0.467	0.046	0.303	0.531	0.068	0.016	0.001	0.001
6	Lh	1.000	0.056	0.091	-0.850	0.851	0.155	-0.301	0.107	0.026	0.188	0.042	0.032
7	Sl	1.000	0.141	0.037	-0.357	0.919	0.068	0.105	0.079	0.008	-0.015	0.002	0.001
8	Sm	1.000	0.056	0.059	0.697	0.889	0.105	0.149	0.041	0.006	0.196	0.070	0.035
9	Sh	1.000	0.014	0.084	1.361	0.593	0.100	-1.112	0.396	0.087	-0.187	0.011	0.008
10	Dl	1.000	0.028	0.033	-0.660	0.701	0.047	-0.228	0.083	0.007	0.366	0.216	0.060
11	Dm	1.000	0.063	0.077	-0.730	0.838	0.129	-0.055	0.005	0.001	-0.316	0.157	0.101
12	Dh	1.000	0.063	0.050	0.505	0.612	0.062	-	0.243	0.032	-	0.145	0.061

								0.318			0.246		
13	Bl	1.000	0.127	0.059	0.378	0.586	0.069	0.307	0.386	0.060	0.083	0.028	0.014
14	Bm	1.000	0.042	0.105	-0.408	0.128	0.027	-0.925	0.658	0.181	0.527	0.214	0.186
15	Bh	1.000	0.042	0.057	-0.533	0.401	0.046	0.181	0.046	0.007	-0.625	0.553	0.263

* co–ordinate to axis

** Co-relation/ Reciprocal contribution

*** contribution

Table 37: Column Contribution.

S.N.	Name	Qual	Mass	Inert	Axis 1			Axis 2			Axis 3		
					Coord	Corr	Contr	Coord	Corr	Contr	Coord	Corr	Contr
1	F 1	1.000	0.070	0.254	-0.921	0.449	0.228	-0.779	0.321	0.215	0.659	0.230	0.487
2	F 2	1.000	0.423	0.169	-0.402	0.770	0.260	0.037	0.007	0.003	-0.216	0.223	0.314
3	F 3	1.000	0.437	0.206	0.373	0.562	0.232	0.298	0.358	0.194	0.141	0.080	0.138
4	F 4	1.000	0.070	0.371	1.020	0.377	0.280	-1.290	0.603	0.588	-0.234	0.020	0.061

Table 38: Analysis of Contingency.

Axis	Inertia	Proportion	Cumulative	Histogram
1	0.2619	0.4999	0.4999	**********
2	0.1991	0.3801	0.8799	********
3	0.0629	0.1201	1.0000	**
Total	0.5239			

Table 39: Correlation Matrix

Characters	F	L	SS	D	S	I	B	A
F	1.00000							
L	-0.90534	1.00000						
SS	-0.10035	0.03231	1.00000					
D	0.58418	-0.60049	0.15689	1.00000				
S	0.65338	-0.46970	-0.22215	0.46011	1.00000			
I	0.17198	-0.04472	-0.33753	-0.40937	0.18818	1.00000		
B	-0.22330	0.22368	0.14022	-0.03449	0.3891	-.11756	1.00000	
A	0.96003	-0.80883	-0.10303	0.54812	0.68498	0.24307	-0.25131	1.00000

Critical value (1 – Tail, 0.5) = + or - 0.30645
Critical value (2 – Tail, 0.5) = +/- 0.36034
N = 30

SUMMARY

Chapter-5

SUMMARY

SLB or maydis leaf blight disease was prevailing in all the block of Jhansi districts. It intensity vary from location to location. The maximum intensity was recorded in Bangra, Mauranipur and Samry villages while minimum in Amababai village of block Baragaon. The maximum conidial length was recorded from the isolates collected from Marry village of Baragaon while minimum length was collected from Baragaon. Similarly, maximum widths of conidia were obtained from isolates of Nemoni village and minimum was from Baragaon. Maximum and Minimum number of septa was recorded from Nemoni and Ambabai village isolates respectively.

Plants infected with SLB, showed extensive large spots with chlorosis. At initiation, the spots were small dot like while after few days it becomes spindle or elliptical in shape with yellow green colour or chlorotic about 2 to 9 mm long and 1 to 2 mm wide. At maturity, spots turn brown to dark brown or black in colour,

targeted like zonate. The spots were parallel to leaf veins. Developed lesions generally occurred singly or coalesced with each other to give a blighted appearance. During severe conditions, lesions were also observed on stem, flower bract and other aerial parts of the plant.

The germination behaviour of conidia at 30°C different incubation period revealed that the maximum hyphal length and number of hyphal cells might be achieved at after 76 h of incubation. Further maximum axial hyphal length (378µ) recorded after 12 days of incubation.

Various characters, governing the disease quality, like no. of leaf/ plant, no. of infected leaf/ plant, no. of spots/ leaf and spot size in different genotypes were also evaluated. Data showed the Average leaf/ plant was 5.8 under pot conditions. Maximum leaf/plant was in Sikkim collection GP-13 and lowest were recorded 4.2 in IG01-534. Average infected leaf/plant was 4.5. Highest infected /plant was in Sikkim collection GP-13 and lowest was in 3.0 in IG01-792. Average number of spots per leaf was 26.3. This was highest in IG01-792 and lowest in African Tall. Average spot size was 4.04 x 1.23 mm. Maximum length of spot was recorded on Sikkim collection GP-13 and Minimum was in IG01-575. Similarly, the maximum width of spot was recorded 2.04 mm and minimum width was 1.0 mm in two-genotype i.e. IG01-788 and IG01-790.

In vitro culture of SLB was found affected by the medium of growth. Colony diameter and morphology of colony were taken as parameters to measure the effect. Maximum growth was observed on czapek's medium and minimum in malt extract. The pathogen growing on Czapek's and Corn meal agar medium produced dark greenish white and superficially well developed colonies with thicker mycelia, whereas on malt extract and oatmeal agar produced light grey to black superficial colonies with thin mycelium. Colonies on Richard's and PDA were dark blackish white poorly branched. Colonies on Czapek's, malt extract and oatmeal agar were deeply submerged. Maximum radial growth (74.8 mm) was observed on Czapek's media at 35°C where as minimum growth was observed in PDA at 40°C (7.0 mm).

Regarding annual recurrence of the disease few studies have successfully been completed through mycelium in plant debris or air borne nature of conidia from distant localities. In case of present investigation, the observations revealed that the role of mycelium in the infected soil and seeds are not possible in Jhansi conditions. The infected soil/seed did not show any disease initiation during experimentation. Survival of inoculum in plant debris and secondary spread through air was confirmed by experimentations. Role of collateral host is another important aspect of disease perpetuation but it requires testing of large number of host species.

Thus it can be concluded that annual recurrence of disease in Jhansi was due to the plant debris/leftover in fields. The can day infestation was due to air.

Attempts were also made to identify the possible source of resistance against SLB. For identification purpose thirty promising genotypes were selected and screened under controlled conditions. The reaction of genotypes was evaluated two ways, One on the basis of disease reaction and secondly on the basis of area under disease progress curve.

On the basis of disease reaction, the genotypes were categorized in seven groups *i.e.* Immune, Highly resistant, Resistant, Moderately resistant, Moderately susceptible, Susceptible and Highly susceptible on the basis of 0-5 point scale.

None of the genotypes was found immune or near immune (Highly resistant to SLB). One genotype i.e. African tall showed resistant. Another genotype J-1006 was found moderately resistant under artificial inoculation conditions. Nine genotypes viz. IG01-535, IG01-728, IG01-674, IG01-804, IG01-792, IG01-799, IG01-678, IG01-806 and IG01-782 were found moderately susceptible. Maximum number of genotypes (16 numbers) proved to be susceptible. And three cultivars i.e. IG01-534, Seti local and IG01 - 691 belong to highly susceptible categories.

According to AUDPC (A-value), the genotypes ranked in eighteen groups. Minimum A-value (most slowly blighted) designated as rank one and maximum A-value (fastest blighted) assigned as rank eighteen. Minimum A-value (94.50) was shown by genotype African tall and maximum A-value (157.50) was shown by IG01-534. Rank 1, 2, 4, 5, 7, 9, 11, 16 and 18 represented by one genotype in each category, whereas two genotypes each were represented categories 3, 6, 8, 10, 13 and 17, and categories 12, 14 and 15 belongs by three genotypes in each group.

Plant pathologists have been concerned primarily with quantifying epidemiological characteristics of patho-systems (Zadoks, 1978). This effort is illustrated by the attention paid to the quality of disease measurement (Large, 1966; James, 1974; Daaman, 1986a, b; Kranz, 1988; Campbell and Madden, 1990 and Gareth Jones, 1998). The ideal disease assessment method should be both precise and accurate (Nutter *et al.*, 1991), as well as reproducible and unbiased, keeping above in mind, present germplasm database includes field assessments using a 0-5 point grading scale based on disease severity and symptom pattern, disease reaction types and quantitative measurements such as infection efficiency, the latent period, sporulation, Basic infection rate and area under disease progress curve to analyze the relationship between semi-quantitative field assessments

and quantitative measurements of monocyclic processes, and then check how these various categories of reaction types correspond to the monocyclic components.

Studies showed that field reaction was dependent on latent period, spot size, sporulation, basic infection rate and area under disease progress curve whereas disease intensity and infection efficiency were independent attributes.

Correspondences analysis was also made to interpret the relationship between rows and columns of contingency table. Analysis showed that all the attributes contribute to the SLB epidemic. Maximum mass *i.e.* 0.148 was occupied by medium latent period followed by low sporulation and low basic infection rate. Column wise, it was maximum in column F3 (Medium to high filed reaction) followed by F2 (Medium field reaction). Maximum inertia was occupied by low latent period (row wise) while column wise it was maximum in F4 (Column represent by high field reaction).

On ordination, most of the attributes placed near axis one. Proportion is about 50 per cent. Thirty eight per cent placed near axis two and 0.120 percent placed near axis three.

Correlations between various characters of database were also made. It is clear from the correlation studies that latent period and sporulation were

negatively correlated with field reaction while other characters are positively correlated with field reactions.

Thus it can be concluded from the present investigation that SLB of maize caused by *D. maydis*, requires detailed study on etiological aspects such as biotype or races. The recurrence of the disease needs more precise investigations every year. Host-resistance/ host parasitic response was also studied. The information generated during the present study can be utilized in disease management. It is well known that the purpose of disease management is to prevent disease damage from exceeding that level where profit significantly diminished. This can be achieved in two ways; firstly either to reduce/delay disease at the beginning of the season or secondly, decrease the rate of disease development during the growing season. This is the first time when various temporal, spatial and genetic attributes of infection process were dealt simultaneously to evaluate host parasitic response in forages. The information generated can be utilized for developing resistant verities of desired traits. African tall and J-1006 can be a good donor as these have high latent period, low sporulation, and infection rate and infection efficiency against SLB pathogen.

REFERENCES

Chapter-6

REFERENCES

Adipala, E.; Lipps, E.P. and Madden, Li V. (1993a). Occurrence of *Exserohilum turcicum* on maize in Uganda. Plant Dis. **77**: 202-205.

Adipala, K., Lipps, P.E., and Madden, L.V. (1993b). Reaction of maize cultivars from uganda of *Exserohilium turcicum.* Phytopathology **83**: 217-223.

Alcorn, J.L. (1983). Generic concepts in *Drechslera, Bipolaris* and *Exserohilum.* Mycotaxon **17**:1-86.

Ali Khan, Shabeer Ahmad; Khan, A. and Ahmad, S. (1992): Influence of temperature regimes and substrate types on colony growth and conidial morphology of *Bipolaris maydis* (Nisik) Shoemaker. Sarhad Journal of Agriculture. **8 (5)**: 551-554.

Ali, K. and Ahmad, S. (1992): Genotype assay of maize for resistance to maydis leaf blight under artificial field epiphytotics of Peshawar region (Pakistan). Sarhad Journal of Agriculture (Pakistan). **8 (5)**: 547-549.

Anonymous (1972). Maize research at pantnagar 1996-72. Govind Ballabh Pant University of Agriculture and Technology, Pantnagar, India.

Anonymous (1987). A handbook of agriculture, P.1303

Aziz, A.; Saleem, M.; Hadayat, U.R.; Rahman and Muhammad; F. (1992): Performance of maize hybrids under irrigated conditions. Sarhad Journal of Agriculture (Pakistan). **8(5)**: 509-512.

Barba, J.T., Reis, E.M., Forcelini, C.A. (2004). Effect of growing substrates on the morphology of *Bipolaris sorokiniana* conidia and the relationship of inoculum density to disease intensity for brown spot of barley. Fitopatologia – Brasileira **29 (1)**:1-6.

Bergquist, R.R. and Masias, O.R. (1974). Physiological specialization *T. turcica f. sp. Zeae* and *T. turcica* f. sp. *Sorghi* in Hawaii, Phytopathology. **6**: 645-649.

Blanco, M.H. and Nelson, R.R. (1972). Relative survival of populations of race T of *H. maydis* on corn hybrids in normal cytoplasm, Plant Dis. Repr. **56**: 889-891.

Botrel D.G. (1979). F.A.O. Plant protection paper and guidelines for integrated control of maize pests. F.A.O. Rome, UN 91.

Byrnes, K.J.; Pataky, J.K. and White, D.G. (1989): Relationships between yield of three maize hybrids and severity of southern leaf blight caused by race 0 of *Bipolaris maydis*. Plant-disease **73 (10)**: 834-840.

Cai, H.W., Gao, Z.S., Yuyama, N., Ogawa, N. (2003). Identification of AFLP markers closely linked to the rhm gene for resistance to southern corn leaf blight in maize by using bulked segregant analysis. Molecular Genetics and Genomics **269(3)**: 299-303.

Campbell, L.C. and Madden, L.V.(1991). "Introduction to Plant Disease Epidemiology". John Wiley, New York.

Carson, M.L. (1995a). A new gene in maize conferring the chlorotic halo reaction to infection by *Exserohilum turcicum*. Plant Dis. **79**: 717-720.

Carson, M.L. (1995b). Inheritance of latent period length in maize infected with *Exserohilum turcicum*. Plant Dis. **79**: 581-585.

Carson, M.L. (1998). Aggressiveness and perennation of isolates of *cochliobolus heterostrophus* from North Carolina. Phytopathology (USA) **88(8)**: 1043-1047.

Carson, M.L., Stuber, C.W., Senior, M.L. (2004). Identification and Mapping of quantitatice trait loci conditioning resistance to sourthern leaf blight caused by *cochliobolus heterostrophus* race 'o' phytopathlogy. **94(8)**: 862-867.

Ceballos, H.; Deutsch, J.A. and Gutierrez, H. (1991). Recurrent selection for resistance to *Exserohilum turcicum* in eight subtropical maize populations. Crop Science **4**: 964-971.

Chang – SeogWon, Hwang – Byungkook (2003). Effects of plant age, leaf position, inoculaum density, and wetness period on *Bipolaris coicis* infection in adlay of differing resistance. Plant disease **87(7)**: 821-826.

Chang, R.Y. and Peterson, P.A. (1995): Genetic control of resistance to *Bipolaris maydis*: One gene or two genes. The Journal of heredity (USA). **86 (2)**:94-97.

Chang, R.Y. and Peterson, P.A. (1995): Resistance to *Bipolaris maydis* is controlled by two genes. Maize Genetics Cooperation Newsletter. **69**: 4-5.

Chaudhuri, S. (1969). Validity of the type of conidial germination as a taxonomic criterion in graminicolous *Helminthosporium* Bull. Indian Phytopathol. Soc. **5**: 15-19.

Chen – XiuRong, Nan – ZhiBiao, Yang-Cheng De, Ren-JiZhou, Liu- Zhaoxiong, Chang – Sheng Hua. (2003). Morphological and biological characteristics of three *Bipolaris* species isolated from grasses in Huanxian grassland, Gansuprovince, Acta – Practaculturae Sinica **12(6)**: 86-92.

CIMMYT. (1994). 1993/94 World Maize Facts and Trends. Mexico, DF.

Collins, N.C., Webb, C.A., Seah, S., Ellis, J.G., Hulbert, S.H., Pryor, A. (1998). The isolation and mapping of disease resistance gene analogs in maize. Mol. Plant microb interact. **11(10)**: 968-978.

Cramer, H.H. (1967). Plant protection and world crop production. *Pflazenschutz Nachrichte,* Leverkusen, Bayer, Germany. 524pp.

Daamen, R.A. (1986a).Measures of disease intensity in powdery mildew (*Erysiphe graminis*) in winter wheat.1. Errors in estimating pustule numbe r. Netherlands Journal of Plant Pathology **92**:197-206.

Daamen, R.A. (1986b). Measures of disease intensity in powdery mildew (*Erysiphe graminis*) in winter wheat.2. relationships and errors of estimation of pustule number, incidence and severity. Netherlands Journal of Plant Pathology **92**:207-222.

Darrah, L.L. (1974). Maize breeding methods study. E.A.F.R.O. Record of Reseach. Annual Report. 65-94.

Darrah, L.L. (1975). Maize breeding methods study, E.A.F.R.O. Record of research Annual Report 60-85.

Darrah, L.L. and Mukuru, S.Z (1976). Maize breeding methods study. E.A.F.R.O. Record research. Annual Report 1976. 54-93.

Darrah, L.L. and Mukuru, S.Z. (1977-1980). Maize breeding methods study. E.A.F.R.O. Record of Research. Annual Report 1977-1980. 123-164.

Degani, O., Maor, R., Hadar, R., Sharon, A., Horwitz, B.A. (2004). Host physiology and pathogenic variation of *Cochliobolus heterostrophus* strains with mutations in the Gprotein alpha subsumt, CGA1. Applied and Enviromental Microbiology, **70(8)**: 5005 – 5009.

Dennis, R.W.G.; Wakefield, E.M. (1946). New or interesting British Fungi. Trans. Br. Mycol. Soc. **29**:141-66.

Dey, S.K.; Dhillon, B.S.; Kanta, U.; Sekhon, S.S.; Saxena, V.K.; Malhi, N.S. and Khehra, A.S. (1993): Resistance to multibiotic stresses in maize (*Zea mays* L.). Journal of Entomological Research. **17(2)**: 75-76.

Dey, S.K.; Mahajan, V.; Pal, S.S.; Dhillon, B.S.; Malhi, N.S.; Khehra, A.S.(1989): Resistance to *Drechslera maydis* in maize inbred lines and their combining ability, Indian Phytopathology. **42(2)**: 261-264.

Dhanju, K.S., Sain-Dass (2005). Evaluation and identification of stable maydis leaf blight disease resistant maize lines and their use in breeding programme. Annals of Agri Bio Research **10(1)**: 39-42.

Dunn, G.M. and Namm, T. (1970). Gene dosage effects on monogenic resistance to northern corn leaf blight, Crop Sci. **10**: 352-354.

Duveiller, E. (1994). A pictorial series of disease assessment keys for bacterial leaf streak of cereals. Plant Dis. **78**: 137-141.

Elliot, C. and Jenkins, M.T. (1946). *Helminthosporium turcicum* leaf blight of corn, Phytopathology 36: 660-666.

Ellis, M.B. (1971). *Dematiaceous Hyphomycetes*. Kew, England: Commonw. Mycol, Inst. 608.

Ellis, M.B. (1976). More *Dematiaceous Hyphomycetes* Kew, England: Commonw Mycol. Inst. 507.

Foroutan, A. and Rahimian, H. (1990): Incidence or corn blight in Mazandaran, Iranian Journal of Plant Pathology. **26 (1-4)**: 120-121.

Gareth Jones, D. (1998). The epidemiology of plant disease. Kluwer Academic Publishers The Netherland. 460p

Garraway, M.O.; Akhtar, M. and Wokoma, E.C.W. (1989): Effect of high temperature stress on peroxidase activity and electrolyte leakage in maize in relation to sporulation of *Bipolaris maydis* race T. Phytopathology (USA). **79 (7)**: 800-805.

Gaunt, R.E. (1995). New technologies in disease measurement and yield loss appraisal. Can. J. Plant Pathol. **17**:185-189.

Gevers, H.O. (1975). A new major gene for resistance to *Helminthosporium turcicum* leaf blight of maize. Plant Dis. Rep. **59**: 296-299.

Gong - guoshu; Zhang - ShiRong (2002). Fractal analysis of lesion patterns of plant disease plant - protection **28(6)**: 9-13.

Gowda, K.T.P.; Gowda, B.J. and Rajasekharaiah, S. (1989): Variability in the incidence of turcicum leaf blight disease of maize in southern Karnataka. Current-Research-University of agricultural Sciences, Bangalore. **18 (8)**: 115-116.

Gowda, K.T.P.; Shetty, H.S.; Gowda, B.J. and Sangam Lal (1993): Incidence of *turcicum* leaf blight of

maize in southern Karnataka. Current Research University of Agricultural Sciences, Bangalore. **22 (6-8)**: 100-101.

Gowda, K.T.P.; Shetty, H.S.; Gowda, B.J.; Sangam, Lal; Sharma, R.C. and Lal, S. (1992): Combining ability and heterosis in maize parental lines for turcicu n leaf blight resistance. Indian Phytopathology. **45(3)**: 321-325.

Green, S., Peng, G., Connolly, T., Boyetchko, S.M. (2004). Effect of moisture and temperature on disease of green foxtail caused by *Drechslera gigantean* and *Pyricularia setariae*. Plant disease **88(6)**: 605-612.

Gupta, M.L.; Janardhanan, K.K. and Akhtar Husain (1978). A leaf blight of casus specious caused by *Drechslera maydis*. Indian Phytopathology. **31(1)**: 243-245.

Hamid, A.H.; Ayers, J.E. and Hill, R.R. (1982). Host x isolates interaction on corn inbreds inoculated with *C. carbonum* race 3. Phytopathology **72**: 1169-1173.

Hanway, J.J. (1963). Groth stages of corn (Zea mays). Agron. J. **55**: 487-492.

Harlapur, S.I.; Mruthunjaya, C.W.; Anahosur, K.H. and Murali Krishna, S (2000): A report survey and surveillance of maize diseases in North Karnataka. Karnataka Journal of Agricultural Science. **13 (3)**: 750-751.

Herbert, T.T. (1982). The rational for the Horsfall Barratt plant disease assessment scale. Phytopathology. **72**:1269.

Hilu, H.M. and Hooker, A.L. (1963). Host pathogen relationship of *Helminthosporium turcicum* in resistant and susceptible corn seedlings. Phytopathology. **54**: 570-575.

Hilu, H.M. and Hooker, A.L. (1965). Localized infection by *Helminthosporium turcicum* on corn leaves. Phytopathology. **55**: 189-192.

Hipskind, J.; Wood, K. and Nicholson, R.L. (1996). Localized stimulation of anthocyanin accumulation and delineation of Pathogen ingress in maize genetically resistant to *Bipolaris maydis* race 0. Physiological and Molecular Plant Pathology. **49 (4)**: 247-256.

Hollier, C.A. and King, S.B. (1985). Effect of temperature and relative humidity on germinability and infectivity of *Puccinia polysora* uredospores. Plant Dis. **69**: 937-939.

Hooker, A.L. (1961). A neaw type of resistance in corn infected with *Helminthosporium turcicum.* Plant Dis. Reptr. **45**: 780-781.

Hooker, A.L. (1963). Inheritance of chlorotic lesion resistance to *Helminthosporium turcicum* in seedling corn. Phytopathology **53**:660-662.

Hooker, A.L. and Perkins, J.L. (1980). *Helminthosporium* leaf blights of corn. The state of the art. Proc. Ann. Corn Sorghum res. Conf. **35**: 68-87.

Hooker, A.L.; Smith, D.R.; Lim, S.F. and Musson, M.D. (1970). Physiological races of *H. maydis* and disease resistance. Plant Dis. Reptr. **54**:1109-1110.

Horner, E.S. (1990): Registration of maize germplasm FS8A (S), FS8A (T), FS8B (S) and FS8B (T). Crop Science. **30(4)**: 964.

Horsfall, J.G. and Barratt, R.W. (1945). An improved grading system for measuring plant disease (abs) Phytopathology, **35**:655.

Hughes, S.J. (1958). Revisiones hyphomycetum aliquot cum appendice de nominibus rejiciendis. Can. J. Bot. **36**: 727-836.

Hughes, S.J. (1980). *Helminthosporium velutinum* fungi Can. No. 163.

Hunt, T.F. (1915). The cereals in America. Orange Judd Co. New York.

Inoue, Y.; Kolnuma, K.; Mochizuki, N.; Kato, A.; (1989): Evaluation of Japanese corn land races belonging to Caribbean flint for southern corn leaf blight (*Bipolaris maydis*) and some other practical traits. Sochi Shikenjo Kenkyu Hokoku. Bulletin of the National Grassland Research Institute. **42**: 49-67.

Iqbal, J. and Ahmad, S. (1994): Assessment of yield loss in maize from maydis leaf blight in Peshawar, Pakistan. Sarhad Journal of Agriculture. **10 (6)**: 703-706.

Ito, S. (1930). On some new ascigerous stages of the species of *Helminthosporium* parasitic on cereals. Proc. Imp. Acad. (Tokyo) **6**:352-55.

James, C.W. (1968). Crop Assessment. In Plant Pathologists Pocket Book, 130-143. CMI. Sec. Ed. Cambrian News Ltd. Queen Street, Aberystwyth wales.

James, W.C. (1974).Assessment of plant disease and losses. Annual Review of Phytopathology **12**:27-48.

Jenns, A.E.; Leonard, K.J. and Moli, R.H. (1982): Variation in the expression of specificity in two maize diseases (fungi). *Euphytica* (Netherlands). **31 (2)**: 269-279.

Jordan, E.G.; Perkins, J.M.; Schall, R.A. and W.L. Pedersen. (1983). Occurrence of race 2 of *Exserohilum turcicum* on corn in the Central United States. Plant Dis. **67**:1163-1165.

Jugenheimer, R.W. (1958). "Hybrid maize breeding and seed production" Agric. Development Paper No. 62. F.A.O., Rome.

Kaiser, S.A.K.M. and Prodhan, H.S. (1990): Evaluation for multiple resistances in maize tc three major foliar diseases. Environment and Ecology. **8 (1A)**: 25-29.

Kaiser, SAKM and Das, S.N. (1989): Occurrence of race T of *Drechslera maydis* (Nisikado) Subram & Jain on maize in West Bengal. Indian Journal of Mycological Research. **27 (2)**: 165-167.

Kamble, H.G.; Sirohi, N.P.S.; Senger, S.M. (2004) improved implements for maize cultivater. Intensive agriculture. IARI Complex, New Delhi-3. XXXXII 07-08.

Kenneth, R. (1958). Contribution to the knowledge of the *Helminthosporium flora* on Gramineae in Israel. Bull. Res. Coun. Isr. Sect. D. **6**:191-208.

Kraja, A.; Dudley, J.W. and White, D.G. (2000): Identification of tropical and temperate maize populations having favorable alleles for disease resistance. Crop Sci. Madison, Wis.: Crop Science Society of America, 1961. **40 (4)**: 948-954.

Kranz, J. (1988). Measuring plant disease. In Experimental Techniques in Plant Disease Epidemiology (J. Kranz and J.Rotem, eds), pp35-50. Springer Verlag, Berlin, Heidelberg, New York.

Lal, B.B. and Chakravarti, B.P. (1976). Assessment of loss due to Brown spot of maize caused by *Physoderma maydis*. Ind. Phytopath **29**: 449-450.

Lam, A. (1984). *Drechslera siccans* from rye grass fields in England and Wales. Trans. Br. Mycol. Soc. **83**:305-11.

Lambert, R.J. and White, D.G. (1997). Disease reaction changes from tandem selection for multiple disease resistance in two maize synthetics. Crop Science (USA). **37 (1)**: 66-69.

Large, E.C. (1965). Measuring plant disease. Ann. Rev. Plant Pathology. 9 (29).

Large, E.C.(1966). Measuring plant disease. Annual Review of Phytopathology. **11**: 47-57.

Leath, S. and Pedersen, W.L. (1986). Effects of the *Ht, Ht1* and/or *Ht3* genes in three maize inbred on quantitative resistance to *Exserohilum turcicum* race 2. Plant Dis. **70**:529-531.

Lee, B.N., Kroke, S., Chou, D.Y.T., Robbertse, B., Yoder, O.C., Turgeon, B.G. (2005). Functional analysis of all nonribosomal peptide synthetases in *cochliobolus heterostrophus* reveals a factor, NPSG, involved in virulence and resistance to oxidative stress. Eukaryolic cell **4(3)**: 545 – 555.

Leonard, K.J. and Suggs, E.G. (1974). *Setosphaeria pralata,* the ascigerous state of *Exserohilum prolatium.* Mycologia **66**: 281-97.

Leonard, K.J.; Levy, Y. and Smith, D.R. (1989). Proposed nomenclature for pathogenic races of *Exserohilum turcicum.* Plant Dis. **79**:779.

Levy, Y. (1989). Analysis of epidemics of northern leaf blight on sweet corn in Israel. Phytopathology **79**: 1243-1245.

Levy, Y. (1991). Variation in fitness among field isolates of Exserohilum turcicum in Israel Plant Dis. **75**: 163-166.

Levy, Y. and Leonard, K.J. (1990). Yield loss in sweet corn in response to defoliation or infection by *Exserohilum turcicum.* J. Phytopathology, **128**: 161-171.

Lipps, P.E. and Hite, R.E. (1982). *Exserohilum turcicum* virulent on corn with the *Ht* resistant gene in Ohio. Plant Dis, **66**: 397-398.

Liu, AiGuo; Zhang, Cheng He; Liu, A.G. and Zhang, C.H. (1997): The developing trend of the predominant population of race 0 of *Bipolaris maydis* and disease resistance monitoring of maize cultivars. Journal of Hebei Agricultural University. **20 (4)**: 35-38.

Liu, K.M.; Su, H.; Cui, Y.; Ma, C.H.; Chen, W.C. and Li, D.L. (1991): Reaction of different male sterile cytoplasm subgroups of the C group maize to the infection of *Bipolaris maydis* race C. Scientia Agricultura Sinica. **24 (4)**: 58-60.

Luttell, E.S. (1957). Leptosphaeria (Metasphaeria) perfect stages of *Helminthosporium turcicum* and *H. rostratum*. Phytopathology **47**:313.

Luttell, E.S. (1963). Taxomomic criteria in *Helminthosporium*. Mycologia, **55**: 643-74.

Luttell, E.S. (1964). Systematics of *Helminthosporium* and related genera. Mycologia **56**: 119-32.

Luttell, E.S. (1976). Ovarian infection of *Sporobolus poiretii* by *Bipolaris ravenelii*. Phytopathology **66:** 260-68.

Luttell, E.S. (1977). Correlations between conidial and ascigerous state characters in *Pyrenophora, Cochliobolus* and *Setosphaeria*. Rev. Mycol. **41**: 271-79.

Luttell, E.S. (1978). Biosystematics of *Helminthosporium*: impact on agriculture. In biosystematics in Agriculture, Beltsville Symposia in Agricultural Research 2, ed. J.A. Romberger, R.H. Foote, L.V. Knutson, P.L. Lentz. 193-209. Montclair: Allanheld, Osmon & Co.

Mahajan, V.; Verma, R.N.; Chandra, S. and Sarma, B.K. (1991): Evaluation of maize (*Zea mays* L,) varieties for resistance to turcicum leaf blight (*Exserohilum*

turcicum) in Meghalaya. Indian Journal of Agricultural Science. **61(7)**: 488-489.

Manwiller, A.; Mmatta, W.K.K. and Ambeta, T. (1985). Breeding hybrid maize at Muguga. Record of Research. KARI Annual Report 61-62.

Matsumoto, S., Noguchi, Y. (1998). Relation between temperature of seeding period and barley leaf stripe disease, caused by *Drechslera graminea.* Bulletin of the Fukuoka Agricultural Research Center (Japan) **17**: 53-56.

Misra, A.P. (1973). *Helminthosporium* species occurring on cereals and other Gramineae. Dholi : Tirhut Coll. Agric. Xii, 289 pp.

Morris, S.W.; Vernooij, B; Titatarn, S.; Starrett, M.; Thomas, S.; Wiltse, C.C.; Frederiksen, R.A.; Bhandhufalek, A; Hulbert, S. and Uknes, S. (1998). Induced resistance responses in maize. Molecular Plant Microbe interactions: MPMI (USA). **11 (7)**: 643-658.

Namjeet Singh; Saxena, V.K. and Singh, N. (1999): Combining ability of early maturing narrow base synthetics for resistance to maydis leaf blight in maize (*Zea mays* L.). Crop Improvement. **26 (1)**: 66-70.

Nelson, R.R. and Tung, G. (1973). The influence of climatic factors on colonization of a susceptible corn hybrid by an isolate of race T. of H. maydis. Plant Dis. Reptr. **57**: 145-148.

Nicholoson, R.L. and Warren, H.L. (1975). Criteria for evaluation of resistance to maize anthracnose. Phytopathology, **66**: 86-66.

Nisikado, Y. (1928). Studies on the *Helminthosporium* diseases of Gramineae in Japan. Spec. Report Ohara Inst. Landwirtsch Forsch. Kurashiki, **4**: 384

Nisikado, Y. (1929). Studies on the *Helminthosporium* diseases of Gramineae in Japan. Ber Ohara Inst. Landwirtsch. Forsch. Kurashiki **4:** 111-26.

Njuguna, J.G.M., Kedera, J.C., Murrithi, L., Songa, S. and Odhiambo, B. (1992). Overview of maize diseases in Kenya. INKARI, Proceedings of a workshop, Review of the National Maize Research Program, KARI/ISNAR Management Training Linkage Project. 1990. 100 pg.

Nutter, F.W. Jr. and Schultz, P.M. (1995). Improving the accuracy and precision of disease assessments: Selection of methods and use of computer-aided training programs. Can. J. Plant Pathol. **17**: 174-184.

Nutter, F.W., Teng, P.S. and Shokes, F.M. (1991). Disease assessment terms and concepts. Plant Disease **75**:1187-1188.

O'Brien, R.D. and Van Bruggen, A.H.C. (1992). Accuracy, precision and correlation to yield loss of disease severity scales for corky root of lettuce. Phytopathology, **82**: 91-96.

Oikawa, T., Ishihara, A., Tanaka, C., Mori, N., Tsuda, M., Iwamura, H. (2004). Accumulation of HDMBOA – GIC is induced by biotic stresses prior to the release of MBOA in maize leaves. Phytochemistry – **65 (22)**: 2995 –3001.

Onderdonk, J.J. and Ketcheson, J.W. (1972). A standardization of terminology for the morphological description of corn seedlings. Can. J. Plant Sci., **52**:1003-1006.

Padmanabhan, S.Y. (1973). The great Bengal Famine. Annu. Rev. Phytopathol. **11**:11-26.

Pal, D. and Kaiser, S.A.K.M. (2001). Effect of agronomic practices on maydis leaf blight disease of maije.

Journal of Mycopathological Research. **39(2)**: 77-82.

Pal, D. and Kaiser, S.A.K.M. (2003). Physical factors that influence the growth and spread of *Drechslera maydis* Nisikado racd 'o' causing maydis leaf blight of maize in India. Journal of Interacademicia (India) **7(3)**: 351 –356.

Pataky, J.K. (1992). Relationship between yield of sweet corn and northern leaf blight caused by *Exserohilum turcicum.* Phytopathology, **82**: 370-375.

Pataky, J.K. (1994). Effects of races 0 and 1 of *Exserohilum turcicum* on sweet corn hybrids differing for *Ht* and partial resistance to northern leaf blight. Plant Dis. **78**: 1189-1193.

Pataky, J.K.; Perkins, J.M. J.M. and Leath, S. (1986). Effects of qualitative and quantitative resistance on the development and spread of northern leaf blight of maize caused by *Exserohilum turcicum* race 1 and 2. Phytopathology **76**: 1349-1352.

Pathak, V.K.; Mathur and Paul Neergaarel (1978). Detection of *Perenospora manshurica* (Naum.) syd.in seeds of soyabean *Glycine mex.* E PPO Bull. 8:21-28.

Paul, A.R. (1971). *Pyrenophora tetrarrhenae* sp. Nov. *Drechslera tetrarrhenae* sp. Nov. and *D. bisepta'a* on Ehrhartege in Australia. Trans. Br. Mycol. Soc. **56**: 261-66.

Payak, M.M. and R.C. Sharma (1978). Research on diseases of maize PL-480 project: Final Technical Report (April 1969- March 1975). Indian Council of Agricultural Research. New Delhi. PP 228.

Payak, M.M. and Sharma, R.C. (1983). Disease rating scales in maize in India. In: Techniques of scoring for resistance to important diseases of maize. All

India Coordinated Maize Improvement Project, Indian Agricultural Research Institute, New Delhi, pp. 1-4.

Pedersen, W.J.; Perkins, J.M.; Radtke, J.A. and Miller, R.J. (1986). Field evaluation of corn inbred and selections for resistance to *Exserohilum turcicum* race 2. Plant Dis. **70**: 376-377.

Pedersen, W.L. and Oldham, M.G. (1992). Effect of three tillage practices on development of northern corn leaf blight *Exserohilum turcicum* under continuous corn. Phytopathology, **76**: 1161-1164.

Perkins, J.M. and Hooker, A.L. (1981). Reactions of eight-four sources of chlorotic lesion resistance in corn to three biotype of *Helminthosporium turcicum*. Plant Dis. **65**: 502-504.

Populer, C. (1978). Changes in host susceptibility with time. Pages 239-262. In plant disease. An advanced treatise. Vol. II. J.G. Hors fall and E.B. Cowlings (eds). Academic Press. N.Y.

Pratt, R.C.; Adipala, E. and Lipps, P.E. (1993). Characterization of race specific resistance to *Exserohilum turcicum* races 0 and 1 in maize Ohso S1 progenies. Plant Dis. **77**: 1227-1232.

Rai, B., Kumar, S., Kumar, B. (2002). Effect of environmental parameters on the development of *turcicum* leaf blight of maize. Annals of Biology **18(2)**: 153-155.

Raymundo, A.D. (1978). Epidemiology of northern corn leaf blight as affected by host resistance and yield losses following simulated epidemics. Ph.D Thesis, University of Illinois, Urbana Champaign. 111 page.

Raziq, F. and Ahmad, S. (1992): comparison of maydis leaf blight epidemics at different stages of maize

development. Sarhad Journal of Agricultu e (Pakistan). **8 (5):** 569-573.

Reddy, S.M. (1975). Conidial germination in some isolates of *Helminthosporium rostratum.* Indian Phytopathol. **28**:285-86.

Reddy, S.M. and Bilgrami, K.S. (1969). Studies on some species of *Helminthosporium.* Indian Phytopathol. **22**:429-37.

Renfro, B.L. and Ullstrup (1976). A comparison of maize disease in Lemperate and tropical environment. Pest Articles and New Summaries. **22**: 491-498.

Robbertse, B., Yoder, O.C., Nguyen, A., Schoch, C.L., Turgco, B.G. (2003). Deletion of all *Cochliobolus heterostrophus* mono functional catalase – encoding genes reveals a role for one in sensitivity to oxidative stress but none with a role in virumlence. Molecular Plant Microbe Interactions **16(11)**: 1013 –1021.

Saghai Maroof, M.A., Van Scoyoc, S.W., Yu, Y.G. and Stromberg E.L. (1993). Gray leaf spot disease of maize rating methodology and inbred line evaluation. Plant dis. **77**: 583-587.

Sain, Das; Basant, Ram; Dhanju, K.S.; Dass, S. and Ram, B. (1992): Inheritance of resistance to *Drechslera maydis* in India. Agricultural Science Digest, Karnal. **12(3)**: 129-131.

Sain, Dass; Dhanja, K.S.; Pawan, Arora; Padam, Singh; Surinder Singh; Yagya Dutt; Dass, S.; Arora, P.; Singh, P.; Singh, S. and Dutt, Y. (2002): Combining ability analysis for resistance to maydis leaf blight (*Drechslera maydis*) disease and yield in maize (*Zea mays*). Indian Journal of Agricultural Sciences. **72(2)**: 125-127.

Sain, Dass; Pawan Arora; Dhanju, K.S.; Surender Kumar; Padam Singh; Dass, S.; Arora, P.; Kumar,

S. and Singh, P. (2000): Gene effects for maydis leaf blight (*Drechslera maydis*) resistance and grain yield in white maize (*Zea mays*). Indian Journal of Agricultural Sciences. **70(5)**: 317-319.

Scharif, G. (1961). Studies on graminicolous species of *Helminthosporium I.H. phlei* (Graham) Comb. Nov. Trans. Br. Mycol. Soc. **44**:217-29.

Scharif, G. (1963). continue (Sic) of studies on Graminicolous species of *Helminthosporium.* Teheran : Ministry of Economy's Press. ii – 97.

Sharma, J.P. and Mishra, B. (1993): Epidemiology and prediction of *turcicum* leaf blight in maize. Indian Phytopathology. **46 (1)**: 81-83.

Sharma, R.C., Rai, S.N., Mukherjee, B.K., Gupta, N.P. (2003). Assessing potential of resistance source for the enhancement of resistance to maydis leaf blight (*Bipolaris maydis*) in maize (*Zea maydis L.*). Indian Journal of Genetics and Plant Breeding **63(1)**: 33-36.

Sharma, R.C.; Payak, M.M.; Shankarlingum, S. and Laxminarayan, C. (1982). A comparison of two method of estimating yield losses in maize by common rust. Lnd. Phytopath. **35 (1)**: 18-20.

Shivankar, S.K. and Shivankar, R.S. (2000). Losses in the grain yield of maize due to *H. turcicum* leaf blight disease. PKU Research Journal **24(2)**: 101-102.

Shoemaker, R.A. (1959). Nomenclature of *Drechslera* and *Bipolaris*, grass parasites segregated from *Helminthosporium.* Can. J. Bot. **37**: 879-87.

Shoemaker, R.A. (1962). *Drechslera* Ito. Can. J. Bot. **40**: 809-36.

Shurtleff, M.C. (1973). Compendium of corn disease. American Phytopathological Society. 105 p.

Singh, M. and Sharma, Y.P. (1976). Evaluation of maize germplasm to banded sclerotial disease and assessment yield loss. Ind. Phytopath. **29**:129-132.

Singulas, K.M.; Hills, R.R. and J.E. Ayers (1988). Genetic analysis of *Exserohilum turcicum* lesion expansion on corn. Phytopathology, **78**: 149-153.

Sivanesan, A. (1985). New species of *Bipolaris*. Trans. Br. Mycol. Soc. **84**:403-21.

Sivanesan, A. (1987). Graminicolous species of *Bipolaris, Curvularia, Drechslera, Exserohilum* and their teleomorphs. CAB Internat. Mycol. Inst. Mycol. Pap. No. 158.

Slopeck, S.W. (1989). An improved method of estimating percent leaf area diseased using a 1 to 5 disease scale. Can. J. Plant Pathol. **11**: 381-387.

Smith, D.R. and Kinsey, J.G. (1980). Further physiologic specialization in *Helminthosporium turcicum*. Plant Dis. **64**: 779-781.

Solomonovich, S.; Levy, Y.; Pataky, J.K. (1992). Yield losses in sweet corn hybrids in response to defoliation and infection by *Exserohilum turcicum*. Phytoparasitica, **20 (2)**: 113-121.

Subramanian, C.V. and Jain, B.L. (1966). A revision of some graminicolous *Helminthosporia*. Curr. Sci. **35**: 352-55.

Thakur, R.P.; Leonard, K.J. and Jones, R.K. (1989). Characterization of a new race of *Exserohilum turcicum* virulent on corn with resistance gene *HtN*. Plant Dis. **73:** 151-155.

Tharmmasak-Sommartya; Prachoom-Jutawantana; Udom-Pupipat. (1995). Disease resistant screening for Northern corn leaf blight. Bankok (Thailand) 13 leaves.

Thind, B.S. and Payak, M.M. (1970). Evaluation of maize germplasm and estimation of losses to Erwinia stalkrot. Plant Dis. Rep. **62**: 319-323.

Tommerlin, J.R. and Howell, T.A. (1988). DISTRAN: A computer program for training people to estimate disease severity on cereal leaves. Plant Dis. **72**: 455-459.

Tsai, W.H.; Tsai, J.N. and Lu, H.S. (1993): Disease development, Meteorological factor and yield losses of sweet corn cultivars caused by southern corn leaf blight. Plant Pathology Bulletin. **2 (1)**: 26-32

Tsukiboshi, T; Koga H. and Uematsu, T. (1996): Races of *Bipolaris maydis* occurring in Japan and their pathogenicity to the *rhm* resistant corn line: JARQ Japan Agricultural Research Quarterly. **30 (2)**: 91-96.

Tsukiboshi, T; Kogo, H. and Uematsul, T. (1995): Identification of pathogenic races of *Bipolaris maydis* occurring in Japan and differences in their aggressiveness. Sochi Shikenjo Kankyu Hokoku. Bulletin of the N.G.R.I. **51**:12-22.

Turner, M.T. and Johnson, E.R. (1980). Race of *Helminthosporium* not controlled by *Ht* genetic resistance in corn in the American corn belt. Plant Dis. **64**: 216-217.

Ullstrup, A.J. (1963). Sources of resistance to northern corn leaf blight. Plant Dis. Reptr. **47**: 107-108.

Ullstrup, A.J. (1970). A comparison of monogenic and polygenic resistance to *H. turcicum* in corn. Phytopathology. **60**: 1597-1599.

Ullstrup, A.J. (1972). the impacts of the southern corn leaf blight of 1970-1971. Ann. Rev. Phytopathology. **10:** 37-50.

Vinh, M.T. and Sarbhoy, A.K. (1991): Amended descriptions of *Drechslera maydis* and

Urohendersonia indica from North-Eastern India. Indian Phytopathology. **44 (2)**: 227-229.

Warren, H.L. (1975). Temperature effects on lesion development and sporulation after infection by races 0 and T of *B. maydis*. Phytopathology **65**: 623-626.

Wastson, G.; Morton, V. and Williams, R. (1990). Standardization of disease assessment and product performance. An industry perspective. Plant Dis. **74**: 401-402.

Wei, J.K.; Lui, K.M.; Chen, J.P.; Luo, P.C. and Lee-Standelmann, O.Y. (1988). Pathological and physiological identification of race C of *Bipolaris maydis* in China. Phytopathology **78**: 550-554.

Welz, H.G. and Geiger, H.H., (2000). Gene for resistance to northern corn leaf blight in diverse maize populations. Plant Breed.: Blackwell Wissenschaft. **119 (1)**: 1-14.

Welz, H.G.; Wagner, R. and Geiger, H.H., (1993). Virulence in *Setosphearia turcica* populations collected from maize in China, Mexico, Uganda and Zambia. Phytopathlogy 83: 1356.

Wilcoxson, R.D.; Skovmand, B. and Atif, A.H., (1975). Evaluation of wheat cultivars for their ability to retard development of stem rust. Ann. Appl. Biol. **80**: 275-281.

Windes, J.M. and Pedersen, W.L., (1990). An isolate of *Exserohilum turcicum* virulent on maize inbred with resistance gene *Ht N*. Plant Dis. **75**:430.

Zadoks,J.C. (1978). Methodology of epidemiological research, in "Plant Disease: An Advanced Treatise", Vol.II (J.G.Horsfall and E.B.Cowling eds), 63-96. Academic Press, New York.